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JOHNS HOPKINS HOSPITAL

REPORTS

VOLUME II

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CONTENTS.

		PAGE.
I.	On Fever of Hepatic Origin, particularly the Intermittent Pyrexia associated with Gallstones. By WILLIAM OSLER, M. D. . .	3
II.	Some remarks on Anomalies of the Uvula, with special reference to Double Uvula. By JOHN N. MACKENZIE, M. D.	32
III.	On Pyrodin. By H. A. LAFLEUR, M. D.	37
IV.	Cases of Post-febrile Insanity. By WILLIAM OSLER, M. D. . .	46
V.	Acute Tuberculosis in an Infant of Four Months. By HARRY TOULMIN, M. D.	51
VI.	Rare Forms of Cardiac Thrombi. By WILLIAM OSLER, M. D. .	56
VII.	Notes on Endocarditis in Phthisis. By WILLIAM OSLER, M. D. .	62
VIII.	Tubercular Peritonitis. By WILLIAM OSLER, M. D.	67
IX.	A Case of Raynaud's Disease. By H. M. THOMAS, M. D. . .	114
X.	Acute Nephritis in Typhoid Fever. By WILLIAM OSLER, M. D. .	119
XI.	The Gynecological Operating Room and the Antiseptic and Aseptic Rules in Force. By HOWARD A. KELLY, M. D.	131
XII.	The Laparotomies performed from October 16, 1889, to March 3, 1890. By HOWARD A. KELLY, M. D., and HUNTER ROBB, M. D. . .	141
XIII.	The Report of the Autopsies in Two Cases Dying in the Gynecological Wards without Operation. By HOWARD A. KELLY, M. D.	167
XIV.	Composite Temperature and Pulse Charts of Forty Cases of Abdominal Section. By HOWARD A. KELLY, M. D.	177
XV.	The Management of the Drainage Tube in Abdominal Surgery. By HUNTER ROBB, M. D.	184

Digitized by Google

PAGE.

XVI. The Gonococcus in Pyosalpinx. By HOWARD A. KELLY, M. D. 195

XVII. Tuberculosis of the Fallopian Tubes and Peritoneum. Ovarian Tumor. By HOWARD A. KELLY, M. D. 201

XVIII. General Gynecological Operations from October 15, 1889, to March 4, 1890. By HOWARD A. KELLY, M. D. 205

XIX. Report of the Urinary Examination of Ninety-one Gynecological Cases. By HOWARD A. KELLY, M. D., and ALBERT A. GHRISKEY, M. D. 216

XX. Ligature of the Trunks of the Uterine and Ovarian Arteries as a Means of Checking Hemorrhage from the Uterus, etc. By HOWARD A. KELLY, M. D. 220

XXI. Carcinoma of the Cervix Uteri in the Negress. By J. WHITRIDGE WILLIAMS, M. D. 224

XXII. Elephantiasis of the Clitoris. By HOWARD A. KELLY, M. D. . 227

XXIII. Myxo-Sarcoma of the Clitoris. By HUNTER ROBB, M. D. . . 231

XXIV. Kolpo-Ureterotomy. Incision of the Ureter through the Vagina, for the Treatment of Ureteral Stricture. By HOWARD A. KELLY, M. D. 234

XXV. Record of Deaths following Gynecological Operations. By HOWARD A. KELLY, M. D. 243

XXVI. The Treatment of Wounds with Especial Reference to the Value of the Blood Clot in the Management of Dead Spaces. By WILLIAM S. HALSTED, M. D. 255

XXVII. A Case of Chorea Insaniens, with a Contribution to the Germ Theory of Chorea. By HENRY J. BERKLEY, M. D. . . 317

XXVIII. Acute Angio-Neurotic Oedema. By CHARLES E. SIMON, M. D. . 339

XXIX. Haematomyelia. By AUGUST HOCH, M. D. 351

XXX. A Case of Cerebro-Spinal Syphilis, with an unusual Lesion in the Spinal Cord. By H. M. THOMAS, M. D. 369

XXXI. Amœbic Dysentery. By WILLIAM T. COUNCILMAN, M. D., and HENRI A. LAFLEUR, M. D. 395

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LIST OF ILLUSTRATIONS.

		PAGE.
1.	Temperature Record,	21
2.	Temperature Record of Dr. Laine's Case,	27
3.	Anomalies of the Uvula (Figs. 1–5),	32
4.	Ground Plan of Private Gynecological Ward,	134
5.	North End of Gynecological Operating Room (No. 1)—phototype,	134
6.	South End of Operating Room (No. 2)—phototype,	135
7.	Composite Pulse and Temperature Chart—26 Normal Cases of Laparotomy,	178
8.	Composite Chart of 10 Normal Laparotomies with High Temperature,	180
9.	Composite Chart of 4 Pus Cases,	182
10.	Drainage Tubes (Figs. 1 and 2),	186–7
11.	Tube Forceps (Fig. 3),	189
12.	Ligature of Trunks of Uterine and Ovarian Arteries (Figs. 1–5),	221–2
13.	Elephantiasis of the Clitoris,	228
14.	Myxo-Sarcoma of the Clitoris (Figs. 1 and 2),	233
15.	Glass Spools (Figs. 1 and 2),	306
16.	Semicircular Instrument Table and Operating Table (Fig. 3),	308
17.	The Operating Table (Fig. 4),	311
18.	The Operating Table with its Perineal Board (Fig. 5),	312
19.	The Yoke of the Perineal Board (Fig. 6),	313
20.	First Dressing after Operation for Carcinoma of the Breast (Plate 1)—phototype,	314
21.	First Dressing after Operation for Inguinal Hernia (Plate 2)—phototype,	314
22.	Various Forms of Intestinal Ulcers in Amœbic Dysentery (Figs. 1–7)—lithograph,	Plate I.
23.	Various Forms of Intestinal Lesions in Amœbic Dysentery (Figs. 1–5)—lithograph,	Plate II.

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24. Various Forms of Ulcers in Amœbic Dysentery (Figs. 1-4)—lithograph, ... Plate III.

25. Ulcers of Large Intestine (Figs. 1 and 2), Liver adjoining Abscess (Figs. 3 and 4)—lithograph, ... Plate IV.

26. Section of Small Abscess of the Liver—lithograph, . . . Plate V.

27. Sections of Chronic Abscess of the Liver and Abscess of the Lung, with Amœbae in Walls of the Abscesses (Figs. 1–4)—lithograph, . Plate VI.

28. Amœbae from Tissues Stained in Various Methods (Figs. 1–15)—colored lithograph, ... Plate VII.

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Volume II No. 1

THE

JOHNS HOPKINS HOSPITAL REPORTS

CONTENTS.

I. On Fever of Hepatic Origin, particularly the Intermittent Pyrexia associated with Gallstones. By WILLIAM OSLER, M. D.
II. Some remarks on Anomalies of the Uvula, with special reference to Double Uvula. By JOHN N. MACKENZIE, M. D.
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VI. Rare Forms of Cardiac Thrombi. By WILLIAM OSLER, M. D.
VII. Note on Endocarditis in Phthisis. By WILLIAM OSLER, M. D.

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JOHNS HOPKINS
HOSPITAL REPORTS.

Vol. II. No. 1.

ON FEVER OF HEPATIC ORIGIN, PARTICULARLY THE INTERMITTENT PYREXIA ASSOCIATED WITH GALLSTONES.

By WILLIAM OSLER, M. D.

Apart from suppuration, we meet with fever as a prominent symptom in the following diseases of the liver: (1) cancer and sarcoma; (2) certain forms of cirrhosis; and (3) chronic obstruction of the bile passages by calculi, tumors, etc.

Cancer.—In all rapidly growing large neoplasms of the liver, there is more or less fever, usually continuous and not often reaching a high grade. Thus of four cases of tolerably extensive cancer of the organ, in which there was fever, in only one did the temperature rise above 102°. In a remarkable case, recently in the Hospital, of cirrhosis with diffuse cancer, the temperature ranged for the first week from 98° to 100°, once reaching 101°, and subsequently remained normal. In the large secondary growths of the organ, the existence of fever when combined with jaundice may be of service, helping to differentiate between this condition and other forms of enlargement of the liver. I have only once seen a marked intermittent fever with severe rigors, in a case of cancer of the liver, and the question of abscess was raised. The post-mortem showed that in addition to extensive secondary cancer, there were several abscesses, caused by the compression of larger gall ducts by the tumors. It is not impossible that a form of intermittent pyrexia might be associated with a rapidly

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growing cancer of this organ, such as Hampeln[1] has described in cases of cancer of the stomach and such as are met with occasionally in rapidly growing lymphatic growths, as noted by Pel of Amsterdam.

Since writing this paragraph, there has been in the Hospital a case of primary cancer of the left lobe of the liver in a young man, who gave a history of several rigors of so pronounced a nature, that among other possibilities that of abscess was discussed.

Cirrhosis.—It is not usually recognized that in cirrhosis there may be fever. The systematic authors on affections of the liver, such as Frerichs and Murchison, speak of slight pyrexia in the early stages, particularly when the organ is enlarged. Carrington[2] has made an interesting study of this question, and states that of forty-four cases in which temperature observations were made, in thirteen fever was a more or less marked symptom. In certain cases, such for example as the one which he described in full, serious difficulties in diagnosis might readily arise and it would be quite pardonable to suspect, from the irregularity of the temperature curve and the existence of slight jaundice, that suppuration was going on. My experience with fever in cirrhosis is small and I have not my clinical records at hand to examine into the point. I have a distinct recollection in one or two instances, particularly in the hypertrophic form, of marked increase in the temperature, but, I should say that in the majority of instances of cirrhosis, fever was not a special symptom.

Obstruction of the Duct by Gallstones.—Since the bile passages have been brought within the sphere of surgery, a renewed interest has been taken in all symptoms which give us more accurate knowledge of the character and situation of lesions in these parts; and I wish particularly in this paper to deal with a form of fever met with chiefly in chronic obstruction of the common duct by gallstones, as it possesses features of the greatest importance for diagnostic purposes. The fever I speak of is intermittent in character and the cases present the following group of symptoms:—

First: Jaundice of varying intensity, deepening after each paroxysm, and which may persist for months or even for years.

Second: Ague-like paroxysms characterized by chill, fever and sweating, after which the jaundice usually becomes more intense.

[1] Zeitschrift f. klin. Medicin, Bd. XIV.

[2] Guy's Hospital Reports, 1884.

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Third: At the time of the paroxysms, pains in the region of the liver, with gastric disturbance.

In a majority of cases this combination of symptoms is, I believe, characteristic of the existence of gallstones in the common duct.

We meet with rigors, fever and sweats in three conditions of the bile passages:—

As an acute and transitory process in ordinary hepatic colic associated with the passage of a stone through the duct.

In chronic obstruction of the duct, usually by stone, without lesions of the bile passages other than dilatation and catarrhal cholangitis.

In suppurative cholangitis produced by gallstones or other causes.

With the first of these, I am not specially concerned, except so far as it may help to explain the occurrence of the paroxysms in the second group. The distinction between the cases of suppurative cholangitis and those of the second category shall be considered subsequently, and I shall now proceed to speak of intermittent hepatic fever with its associated symptoms as characteristic of chronic obstruction of the duct by gallstones and without suppuration.

The literature of the subject, though interesting, need not, for the purposes of this paper, be discussed at length. Of the numerous writers on gallstones during the last century, Soemmering[1] appears to be the only one to mention the symptom, using the phrase, in speaking of the fever associated with gallstones, "et ipsa febris intermittens."

We owe to French physicians our knowledge of this valuable symptom. Monneret[2] is usually credited with its recognition, but the thesis of Magnin[3] and the work of Charcot[4] present us with the first satisfactory studies, from which indeed has been derived most of the information on the subject which we find scattered through the text-books and monographs.

Among German writers the work of Frerichs contains many cases, illustrating this symptom of chronic obstruction, but he does not appear to lay special stress upon its importance in diagnosis. In von Schueppel's article upon gallstones, in Ziemssen's Cyclopædia,[5] the remarks are based on the work of the French writers. Wagner[6] has reported interesting cases. References to these symptoms occur in

[1] De Concrementis biliariis, 1795.
[2] Pathologie Interne, Tome I.
[3] Paris, 1869.
[4] Leçons sur les Maladies du Foie, 1877.
[5] Vol. IX.
[6] Deutsches Archiv. f. klin. Med., Bd. XXXIV.

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the various German text-books, but the question does not appear to have received the full consideration which its importance demands, and the majority of the writers, as Strümpell, for example, speak of the *fièvre intermittente hepatique* as if it were always associated with suppuration.

Among English writers, Murchison, in his work upon the liver (third edition), notes the occurrence of rigors in chronic obstruction, and in his paper upon conditions causing an intermittent fever,[1] he deals more fully with the general features of the affection. Harley, in his work on the liver, does not mention it. Ord refers to it in his paper on some of the rarer symptoms produced by gallstone.[2] In the English text-books on medicine, it is not often spoken of; even Fagge, whose work is such a store-house of clinical facts, has no reference to the subject.

In this country, the question has been discussed by Bartholow, who gives, in Pepper's System of Medicine, a full summary of the French observations. In Sajous' Annual for 1888, Dr. W. H. Thompson, of New York, refers to intermittent hepatic fever as occurring frequently in this country and as well recognized by authors; but in a private communication he informs me that he had been under a misapprehension, and so far as he knows the subject had not been discussed by any American writer. Musser,[3] of Philadelphia, has reported several interesting cases.

The following cases have been under my observation :—

CASE I.—*Jaundice of three years duration. Repeated attacks of chills and fever; cholæmia, death. Gallstones in common duct.*

J. H. R., aet. 68, admitted to the Johns Hopkins Hospital, May 25th, 1889, complaining of jaundice, chills and fever, which had lasted on and off for three years. With the exception of attacks of eczema, he had been a healthy man until three years ago, when his present trouble began with dyspepsia and pain in the pit of the stomach. In the first attack there was sharp pain in the epigastrium, followed by a chill and vomiting. These recurred very frequently, and with them he invariably became deeply jaundiced and the stools

[1] Lancet, 1879. [2] British Medical Journal, 1887, I.

[3] On Paroxysmal Fever, not Malarial. Proceedings of the Phila. Co. Med. Society, 1884.

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were putty colored. He had been subject to catarrh of the stomach and had always been constipated. The attacks of chills and fever had, at times, been very severe, and he would sweat heavily after them. On the occasion of his first visit to Hospital a violent attack came on while he was in the waiting-room; he shook as in an ague paroxysm. His wife stated that he had rarely passed three weeks without a chill of great severity.

Present condition.—Much emaciation; skin dry and harsh and of an intensely bronze color. It presented many small scabs, the result of scratching. The muscles were very flabby. The conjunctivæ and mucous membrane of the mouth were stained, as were also the nails of the fingers and toes. The expression of the face was dull and the speech slow. Articulation was impaired from dryness of the mouth. Examination of the thoracic organs negative. Pulse 68, small and regular. Abdomen was a little distended, somewhat tympanitic, everywhere painless.

The edge of the liver could not be felt. Dullness in mammary line began at the 6th rib and extended 2½ inches (6+ cm.) vertically. The most careful palpation could not discover the gall bladder. The splenic dullness was slightly increased, but the edge was not palpable. The urine was of a deep brownish-red color, acid in reaction, specific gravity 1008. It contained a small amount of albumen and a few tube casts. Temperature on admission was 98°. His chief complaints were of intense itching of skin and of occasional pains in the abdomen.

On the morning of the 26th he had a chill, in which the temperature rose to 101° and he became delirious, would not answer questions, and wanted constantly to get out of bed. The temperature sank to about 96° and remained at that point until eleven o'clock when it rose to 97°. Gradually coma supervened; the pulse rate increased to 160 and the respiration became very irregular, 30 per minute. He was given an active purge and sweated. The coma gradually deepened; the temperature rose, reaching towards evening 101°. He died early on the morning of the 27th.

From his history and the repeated attacks of hepatic intermittent fever extending over a period of three years (a period of sufficient length to exclude suppurative cholangitis, abscess, or cancer), I made a diagnosis of obstruction of the common duct by gallstones, and suggested to him the propriety of an operation. To this he had

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given his consent, and entered the Hospital with a view of remaining a week to gain strength before submitting to it.

Post-Mortem, by Dr. Welch. Peritoneum contained 30 cc. of yellow serum.

In thorax the pleural membranes were normal. The pericardium contained an excess of fluid. The heart was a little enlarged, weighing 11½ oz. (326+ gms.) The segments of the aortic valve were indurated along free and attached margins. On the aortic aspect of one segment was a fresh, reddish-grey, partially detached, vegetation. The remaining two segments had coalesced in consequence of ulceration and nearly total disappearance of the septum, in the situation of which was an irregularly indurated, ulcerated, slightly elevated ridge, partially covered with red-grey fresh vegetations. On the ventricular aspect of this fused segment was a vegetation 15x10 mm. The mitral and other valves were normal. The heart muscle was flabby and brownish in color; on microscopical examination not fatty.

The lungs were normal.

The spleen was 15 cm. long and 10 cm. in breadth. Its contents were soft and dark red in color.

The combined weight of the kidneys was about 12 oz. (340+ gms.) Cortex of average thickness. The striæ obscured; organs not firm.

Bile duct. The orifice of the common duct was dilated and contained a plug of thin, pale yellowish mucus, easily displaced. Upon passing a probe into the duct it entered a sac 1½ cm. from the orifice, which corresponded to the dilated ductus communis choledochus. A round ulcerated opening 3 mm. in diameter communicated between the lumen of the duodenum and the common duct in its course in the intestinal wall. A sac resulting from the dilatation of the common duct measuring 2½ cm. in diameter was completely filled with gallstones of varying sizes, the largest being 2 cm. long by 1½ thick; the smallest not larger than a pea. All were provided with facets. The walls of the dilated common duct were thickened and the surrounding connective tissue very dense and intimately adherent to the adjacent parts. The gall bladder was shrunken to a small sac 2 cm. in length by 2 cm. in breadth. The walls were thickened and it contained a number of gallstones around which it had contracted. The cystic and hepatic ducts were greatly dilated and contained gall-

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stones; altogether not less than twenty could be felt in the duct. Externally they were deep black in color.

The liver weighed 3 lbs. 11½ oz. (1686.8 gms.); the surface was smooth, mottled greenish and pale yellow in color. Outlines of the lobules were indistinct.

On section the bile ducts appeared moderately dilated. The walls were thickened and the contents yellowish, viscid, and not purulent.

The aorta presented several small atheromatous ulcers.

CASE II.—*History of previous attacks of gallstone colic. For eight months recurring attacks of pain with ague-like paroxysms and intensification of the jaundice. Passage of the gallstone. Recovery.*

N. K., aet. 30, a dark, slightly built woman, was admitted to the Montreal General Hospital November 17, 1879. She had been healthy with the exception of occasional attacks of indigestion. Four years before she had several attacks of cramps in the abdomen. In the middle of September, 1879, they recurred after a wetting. At this time she had vomiting, and the attacks were of such severity that morphia was given hypodermically. Two days after the onset she became deeply jaundiced, the attacks of pain recurred, and the vomiting became very troublesome, but in about two weeks she was able to go to her home, where she remained until November 17th. When admitted she was deeply jaundiced, the tongue was furred, she had nausea and looked feeble. She remained in hospital during the winter, and I found her in Ward 23 when I went on duty. During a residence of five and one-half months in hospital her chief symptoms had been: first, jaundice, varying greatly in intensity, sometimes almost disappearing, but recurring again in a few days; second, ague-like paroxysms—chills, fever and sweating—accompanied by severe abdominal pains, coming on at intervals of from three to ten days; third, great impairment of appetite, dyspepsia, frequent vomiting, especially about the time of the paroxysms; fourth, great tenderness in the epigastrium, most marked over the right costal border.

After an interval of a week or ten days—during which the jaundice would diminish, the bile almost entirely disappear from the urine, the fæces become slightly bile-tinged, the appetite improve, and the patient would sit up—the paroxysm would come on, either with a slight chill, not more perhaps than a transitory feeling of cold; at

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others it would be a severe rigor, in which she would shake as if in an ague-fit. This cold stage lasted from fifteen minutes to three or four hours and was followed by great heat of the skin and burning fever, which after continuing for an hour or two would be followed by a profuse perspiration. The temperature, which was usually normal, or even sub-normal, would rise in the attacks, reaching 102°–104°, subsiding quickly after the paroxysm, and sometimes sinking to 97°. The fever rarely persisted for an entire day. Among the concomitant symptoms of these attacks, vomiting with severe gastric pain were the most common. The pain which usually gave indication of the onset resembled that of hepatic colic, being epigastric, and radiating to a point beneath the right shoulder blade. It was scarcely ever as agonizing as the pain of ordinary biliary colic, but was often severe enough to require morphia. Before and after the attacks the epigastrium was very tender, so much so that she would even complain of the weight of the bed clothes. Vomiting was a marked feature, and usually accompanied the paroxysm. The bowels were moved every day, sometimes two or three motions. The color of the fæces depended on the intensity of the jaundice—light color when the skin was deeply tinted; brownish when the color of the skin was less intense.

For a long time the motions were filtered in the hopes of finding gallstones. Invariably after an attack the jaundice deepened, and we could generally tell by her appearance alone whether she had had one. The urine also at this time became deeply bile-tinged. In the intervals the pain subsided, and the nausea and vomiting became less troublesome, but for days she could not take anything but a little biscuit and milk. She usually remained in bed, but during a long interval she would get up and go about the ward. Itching of the skin was occasionally a distressing symptom.

In April I made the following notes:

"Moderate jaundice; nothing special to be seen on inspection of the abdomen; on palpation decided tenderness in the epigastric region, most marked towards the right costal border; no fullness or increased resistance; limit of dullness extends in nipple line from upper border of 6th rib to within half inch (1.3 cm.) of the margin of the rib; splenic dullness 2½ inches (6.3 cm.); heart and lungs normal; urine bile-tinged, specific gravity 1020, no albumen; enormous dark,

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granular, bile-stained casts, some containing epithelial cells; fæces clay-colored, soft and a little offensive; no fever."

Towards the end of April she left the hospital and went to her home at St. Johns, where she was attended by Dr. Robert Howard, who treated her for gallstones, giving large doses of bi-carbonate of potash. She had several paroxysms of pain with fever, and the jaundice continued. On June 3rd she passed per anum a large round gallstone, which Dr. Howard kindly sent to me. It weighed 60 grains (3.9 gms.), and measured a little over one cm. in diameter. She improved very rapidly after this, the jaundice disappeared, and she recovered her usual health and strength.

CASE III.—*From July, 1879, until August, 1882, jaundice of varying intensity, with recurring attacks of pain and intermittent fever. Recovery.*

November 9, 1880, I was asked to see Mrs. S., aet. 55, a well-nourished woman, wife of a florist. She had always been healthy and had borne five children. Had been accustomed to work in the garden and in the greenhouses. Her illness began July, 1879, and her physician, Dr. Simpson, gave me the following particulars of the onset and development of the disease.

"In July, 1879, Mrs. S. consulted me at her house for a mild attack of jaundice, which she ascribed to having seen a disgusting object which emitted a most offensive odor, causing her to feel sick at her stomach. When a young girl she had an attack of jaundice following a fright. On August 4th, I saw her again; the jaundice had deepened and she complained of a dull pain in the region of the liver and general distress. She remained in this state until the morning of the 6th, when she was seized with a severe chill and intense pain below the ribs on the right side, extending into the epigastrium and to the right shoulder. It was increased by pressure and motion, the breathing was hurried and the anxiety of the patient most distressing. A chill of about two hours was followed by high fever, then copious sweating, which stained the sheets a deep yellow color. The liver was distinctly enlarged. The pain gradually abated but the tenderness persisted for several days. All the essential phenomena of jaundice were present. She remained under my care until January, and during this time she had a paroxysm every two or three weeks, varying somewhat in in-

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tensity and duration. The pain gradually became less and less. The chill, fever and sweating were invariably present after each attack, and the jaundice deepened. Itching of the skin was a most distressing symptom, preventing sleep and rendering life almost unendurable. For days at a time the stools were strained, but without finding gall-stones. The enlargement of the liver disappeared."

During the early part of the year the attacks continued, but during the summer, under homœopathic treatment, the jaundice almost disappeared, and for many weeks she had not a paroxysm. When first I saw her she was intensely jaundiced and suffered with the most terrible itching of the skin which I have ever witnessed. Warm alkaline baths were ordered with great benefit. One night after a bath she became quite incoherent. On examination, her condition was as follows: well-nourished, somewhat stout woman; thick layer of panniculus over the abdomen. She says, however, that she has lost flesh during the past year. The skin has a deep greenish-yellow tint, and is covered with scratches; edge of the liver could not be felt; no tumor evident below the right costal border; she winces when firm pressure is made between the navel and costal margin; area of liver dullness somewhat diminished and the organ is not tender to firm pressure; the splenic dullness is increased, 7 inches in vertical diameter (17.8 cm.); heart and lungs normal; tongue red and indented with the teeth; bowels irregular; stools clay-colored and offensive; urine very dark-colored and contained much bile pigment; temperature 98.4°; appetite poor, can only take soft food. Within a few days the itching disappeared, excepting on the palms of the hands and the soles of the feet. These parts had always been the most troublesome, and the pads at the bases of the fingers were much swollen and tender. By the 15th she was very much better. The jaundice had begun to disappear, but at noon on the 16th she had a very severe paroxysm, the chill lasting nearly two hours, and there was no vomiting with this attack and no special abdominal pain; no change noticed in the hepatic region.

From this time until Christmas day she had seven severe attacks, varying in intensity, five of which followed each other on Fridays. The rigors were most intense in violence, shaking the bed and causing the room to vibrate. Temperature reached from 103° to 104°. The jaundice intensified after each attack.

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After Christmas she improved very much; jaundice almost entirely disappeared and she was able to get up and go about the house. On two occasions she had severe headache and great depression, followed by copious sweating. The palms of the hand continued very tender. A troublesome symptom was the profuse sweating about the waist, sufficient to saturate the under-linen and render it necessary to wear cloths about her. The urine became clear, the fæces contained bile; the liver showed no special alteration. The tenderness on the right side of the epigastrium persisted. During the spring of 1881 the daily amount of the urea was estimated during a period of three weeks, but there was no special diminution during the paroxysms.

I lost sight of Mrs. S. after the spring of 1881, when she was still considerably jaundiced and had paroxysms at prolonged intervals.

On the 7th of July, 1882, she came to see me and stated that her condition had remained unchanged; the paroxysms still recurred at intervals, but she once passed six weeks without one. In May of this year she had them worse than ever, and to use her own expression, "she was dead of them." After August, 1882, the jaundice disappeared and she now looks in perfect health.

Dr. F. G. Finley, of Montreal, recently (Oct. 1888) made inquiries for me about this patient and writes that she continues well and has had no return of the pain or of the jaundice.

CASE IV.—*Repeated attacks of biliary colic. For three months jaundice with repeated paroxysms, chills, fever and sweats. Operation. Death. Gallstone in common duct.*

Mrs. S., aet. 51, patient of Dr. Bolling, of Chestnut Hill. Seen March 2d, 1887.

She had been a healthy woman, but since 1862 had several attacks of biliary colic, on one occasion with jaundice. Since Christmas she she had pain in the upper part of the abdomen, and very severe jaundice, which has gradually deepened. The urine has been intensely bile-tinged and the fæces clay-colored. For two weeks she had been worse and confined to bed. A special feature had been chills, recurring daily, followed by fever rising to 103° and 104°, and then copious sweating. The chills were most severe and the fever most pungent. The stools had been carefully examined for gallstones, but without result. The patient was a well-built, well-nourished woman, with

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intense icterus; tongue coated and dry; pulse 120, small and feeble; no fever; abdomen large; fat abundant; liver dullness not increased. On palpation, nothing to be detected along the costal border in the right hypochondrium; towards the epigastrium great tenderness and distinct sense of increased resistance. A most careful examination failed to reveal the presence of enlargement of the gall bladder. The history of the previous attacks, the persistency of the present one, and the recurrence of intermittent fever pointed clearly to obstruction of the ducts, probably by gallstones. The question of surgical interference was raised, and possible obstruction by malignant disease at the head of the pancreas was also debated.

March 3d. The patient was seen at 2 p. m. by Doctors Agnew, J. W. White, and Bolling. The condition was worse. The patient was weaker; tongue very dry; abdomen distended; diffuse tenderness, and in the epigastrium extreme sensitiveness to pressure.

Dr. Agnew made an incision between six and seven inches (16 cm.) in length, the outer edge meeting the rectus muscle. When the peritoneal cavity was opened a bile-tinged, slightly turbid fluid escaped. The liver looked very dark, and a conical, pointed gall-bladder projected beyond the edge not more than one inch (2.5 cm.) from the surface, the liver being slightly atrophied above it. On lifting the liver the bladder was seen to be enormously dilated, and by aspiration 18 oz. (431 gms.) of dark bile were removed. There were no gallstones in it, but a stone was felt low down in the common duct and pushed back into the gall bladder and removed. The head of the pancreas seemed hard and indurated but not enlarged. The patient sank and died twelve hours after the operation. No autopsy was allowed.

CASE V.—*Jaundice of two and a half years duration. Recurring attacks of intermittent fever, with pains. Operation. Death. Gallstone in the common duct.*

A. B., a woman aged 40, was in the Philadelphia Hospital, September, 1887, when I took charge of the wards. She had been under my care previously, in the spring of 1887, when I was on duty for Dr. Tyson. This had been her third or fourth admission within two years with attacks of pain in the region of the liver, and chills, fever and heavy sweats. My colleagues had on two occasions brought her blood to me for examination, the existence of malaria having been

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suspected. Once certainly, possibly twice, her liver was aspirated, the recurring chills having aroused a suspicion of abscess.

The patient was a medium-sized, fairly well-nourished woman. She had lived a hard life and had had specific disease. Attacks similar to those from which she at present suffered, came on about two years ago and she had not been entirely free from them for a period of three months, nor does she think that she had in this time ever passed two months without a slight tinge of jaundice. When first seen, she was up and about the ward and showed only the slightest lemon-tint of the skin and of the conjunctivae. The urine was a little high-colored. The stools contained bile. On examination the liver appeared to be enlarged. In the mammary line, the right lobe extended four fingers breadth below the costal margin; in the median line a distinct irregularity in outline could be made out. The gall-bladder could not be felt. Palpation was not painful. Early in October, she had an attack of violent pain with vomiting and a moderately severe rigor, after which the temperature rose to nearly 104°, and she sweated profusely, the entire paroxysms lasting over twelve hours. The next day she was distinctly jaundiced, free from fever, the tongue heavily coated and the stomach extremely irritable. The urine was very dark, containing bile-pigment and the stools were light-colored. The liver did not seem to be larger but it was sensitive to pressure. The gall-bladder could not be felt.

In three or four days, the gastric symptoms passed away and she was able to sit up. The jaundice deepened distinctly for three or four days and then gradually lightened.

The case was made a subject of almost daily demonstration in the ward-class and I confidently predicted a return of the paroxysms. Throughout the winter she had four or five, each similar to the one just described, varying somewhat, however, in intensity.

I had made up my mind from the length of time which the woman had suffered and from the character of the attacks that the case was one of obstruction of the common duct by gallstone; and early in February, I asked Dr. White to see her in consultation. The patient consented to an operation, and Dr. White made a free abdominal incision along the line of the costal cartilages. There was extensive perihepatitis with puckering of the edges of the liver, due to the cicatrization of old gummata. The gall-bladder was not enlarged;

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there was a great deal of fibroid matting of the tissues in the gastro-hepatic omentum. No gallstone could be felt in the gall-bladder, nor in the duct. The patient came out well from under the influence of ether; had no shock, and six hours afterwards her temperature and pulse were normal. The following day there was a rise of temperature and she died on the third day after the operation.

I had been so confident, from the history of the case, that it was one of obstruction by gallstones, that I was naturally chagrined at the negative result of the operation. The friends removed the body at once to Jenkintown, but I was fortunately able to secure an autopsy, when the following condition was found.

Perihepatitis with deep puckering, owing to the cicatrization of old gummata. The liver was not enlarged; the apparent increase in size, during life, was due to the tilting forward of the convex surface of the organ. There was recent acute peritonitis, confined to the region above the transverse colon. The liver, stomach and duodenum were removed together for dissection. On slitting open the duodenum, a bile-tinged mucus was seen oozing from the papilla. Projecting into the duodenum and covered by the mucosa only was a gallstone, the size of a marble. It lay entirely within the bowel, quite close to the narrow orifice of the duct, through which it could be seen after the removal of the mucus. The stone could not be moved up or down, though it had slight play in the dilated pouch, at the termination of the duct. The common duct and its main branches were dilated; the former about the size of the index finger. The contents of the duct was a bile-stained mucoid fluid. The cystic duct was wide. The gall-bladder was a little enlarged, but did not contain any stones. The terminal bile-ducts were not dilated. The other organs presented no special change.

CASE VI.—*Jaundice of varying intensity from July, 1887, until August, 1888. Repeated paroxysms of intermittent fever. Death.*

A. B., aet. 70, physician. Family history good; has enjoyed excellent health with the exception of an attack of nervous prostration in 1863. Some years after he got stout and was unable to take proper exercise. He never had a strong digestion and always had to be careful in his diet. He was in his usual health until July, 1887, when he had an attack of jaundice, coming on with severe pain, evi-

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dently biliary colic. The jaundice gradually disappeared, but returned in five or six weeks with pains of the same character. The second attack did not last so long, but in December he had a third attack, again associated with pain in the upper portion of the abdomen. Since then he has not been free from the jaundice, which has, however, varied greatly in intensity. About Christmas he had a severe chill, followed by fever and sweats. At intervals of about ten days the paroxysms have recurred, and after each one the jaundice deepened. On March 10th, when I saw the patient with Dr. Murray Cheston, his condition was as follows: Stout, well-nourished old man; deeply jaundiced; pulse 92, feeble; no fever; skin moist; complained of much itching; tongue coated; abdomen large; panniculus thick; omental fat excessive. On palpation, the edge of the liver not to be felt; dullness diminished, not more than three fingers breadth in the middle line, and two and one-half inches (6.35 cm.) in the nipple line; no tenderness on deepest pressure over the pancreas. The patient was bright mentally. No vomiting had occurred throughout the illness, but there had been attacks of nausea; bowels constipated; stools of the consistency and color of putty. They have not been dark and normal in appearance for months. Urine scanty, deeply bile-tinged.

The night before I saw him he had a very severe chill, lasting one and one-half hours, which was followed by a burning fever and profuse sweating. He was much prostrated by the attack, and in the morning, as was usual after an attack, the jaundice had deepened and intensified. He was not emaciated, though he said that he had lost flesh, particularly in the limbs, during the past three months. I saw him at intervals of a few weeks for several months. Throughout April he had no chills and was comparatively comfortable, and the jaundice began to lighten. In May he had several very severe paroxysms, in which the temperature reached 103°–104°. After each one the color became more intensified, and the urine became darker. With some of the chills he had severe abdominal pains, but with others he complained only of a sensation of epigastric distress. Throughout the summer the chills and fever persisted at irregular intervals. In August the jaundice deepened and he died comatose. No autopsy.

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CASE VII.—*Jaundice, with attacks of colic, of ten (?) years' duration. Under observation for three years, with repeated attacks of intermittent fever, always associated with an increase in the jaundice.*

A. B., aged 46, single, domestic by occupation, was admitted to the Philadelphia Hospital with fever and jaundice.

There was nothing special in the family history. Ten years ago she had the first attack of jaundice, which came on with pain in the abdomen, particularly on the right side. She was in bed for two weeks. From that date until the present the skin has never been of the normal color, though for weeks the jaundice would be extremely light. During this period she has had repeated attacks of pain in the region of the liver, usually accompanied with vomiting and diarrhœa. In one of these "spells," as she calls them, she was admitted to Hospital. She states that for the past ten years she has had on an average three or four of these attacks a year, always associated with chills and fever and with sweats. She has had also what she terms "burning spells," in which she would get very hot but would not sweat.

Inspection. Patient not emaciated. There is a thick layer of fat over the abdomen; the face is fairly plump; she is deeply jaundiced, color of dark, olive-yellow, not the light soft tint of recent icterus. The conjunctivæ are deeply stained. The skin is dry and harsh. There is no eruption, only a few scratches on the back. She complains of intolerable itching. Temperature was 103° on admission, but fell to the normal; pulse 100. The abdomen is symmetrical, the upper zone not especially enlarged. On palpation it was soft, non-resistent and painless until the epigastric and right hypochondriac regions were reached, which on pressure were extremely tender. The edge of the liver can be felt just below the costal margin. The gall-bladder is not palpable.

Percussion in mid-sternal line shows not more than two inches (5 cm.) of liver dullness; in nipple line about three inches (7.6 cm.).

The spleen is not palpable. There are three inches of vertical dullness in the axillary line.

She has had several movements of the bowels since admission; the fæces are soft and of a grayish-brown color. The urine is high-colored and contains bile-pigment, no albumen.

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This patient was under my care on three separate occasions during three years. Each time she was admitted with fever and great pain in the epigastric region, with vomiting and diarrhœa. These attacks usually set in with a heavy chill. The jaundice would gradually get a little lighter, but never completely disappeared. The stools were never quite clay-colored. I urged her repeatedly to submit to an operation, but she would never consent. Twice she was made the subject of a clinic illustrating a form of hepatic intermittent fever, due in all probability to chronic obstruction by gallstones.

CASE VIII.—*Jaundice of ten months duration, with recurring chills and fever. Recovery.*

Agnes S., aged 23, was admitted to the maternity wards of the Philadelphia Hospital in January, 1886. Her labor was normal and the convalescence uninterrupted. Two months after confinement, she fell across a chair and injured herself severely, causing a profuse hemorrhage, said to have been uterine. Three days after the accident she had nausea and vomiting, and in the course of a week jaundice developed. When admitted to the medical ward, she was slightly yellow and complained of pain in the epigastrium and of back-ache. The bowels were constipated and the stools of a clay color; they were frequently examined for gallstones, but none were found. A few weeks after the onset of the jaundice—the exact date is not stated in the notes—she began to have febrile attacks, preceded by a chill and followed by profuse sweating. These attacks recurred at irregular intervals. I saw her first in August, in the obstetrical department, and it was then thought that she had either malaria or abscess of the liver. She was repeatedly made the subject of ward-class demonstration as an instance of true hepatic intermittent fever, probably depending upon gallstones obstructing the common duct. Examination of the liver was negative; the edge could be distinctly felt. The gall-bladder did not seem to be enlarged. There were three inches (7.6 cm.) of vertical liver dullness in the nipple line and the same in the mid-sternal line. The spleen was not palpable. Throughout August, she had four severe chills; after each one the jaundice deepened and each was accompanied by nausea, vomiting and a good deal of pain. In September the paroxysms were less frequent, but she had two distinct rigors, on the 3d and on the 13th. On the 25th, the temperature rose to nearly

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102°, and she had, for ten days, an irregular intermittent fever. On the 2d, 3d and 4th, there were chills, and she had become at this time more intensely jaundiced than at any period since her admission. She improved between the 5th and the 15th, and the jaundice got lighter. On the latter date she had a heavy chill. Chills recurred on the 22d, 26th and the 28th. In the first twelve days of November she had six rigors; the jaundice again became deeper. After this date she improved very much and through the latter part of November and the early part of December, she was remarkably well and presented only a light-lemon tint. She had two or three slight chills, each followed by an increase in the jaundice. On the 27th and 28th, the paroxysms recurred and she again became jaundiced. After January 1st, the color became lighter, and by the 18th, when she went out, the jaundice had almost disappeared. I saw her again more than three months afterwards and the jaundice had completely disappeared and she had had no recurrence of the attacks.

The accompanying temperature record of Case VIII (*see p. 21*) illustrates very well the type of fever met with in these cases.

Of these eight cases, six were women. Two died after operation; two died from the effects of the long continued jaundice; three recovered after the persistence of the condition for from eight months to three years, and one passed from observation.

In analysing the symptoms associated with these paroxysms, we have—

First: *Jaundice.* This was present in every instance and may be said to have been constant, though varying very greatly in its intensity. It will have been noticed that in every one of the cases the statement occurs that after the paroxysm the jaundice invariably deepened. I do not remember ever to have seen a well-marked paroxysm, with intense rigor and high fever, in which this peculiarity did not occur. The patients soon learned to recognize it and to expect, as a matter of course, an intensification of the jaundice. With this, the amount of bile-pigment increased in the urine and the stools became more clay-colored. After persisting for a week or ten days, the tint would become lighter, until as in Cases II and VIII, the skin would become, in the intervals, almost normal. The urine, too, would be lighter in color and the stools contain bile. In certain

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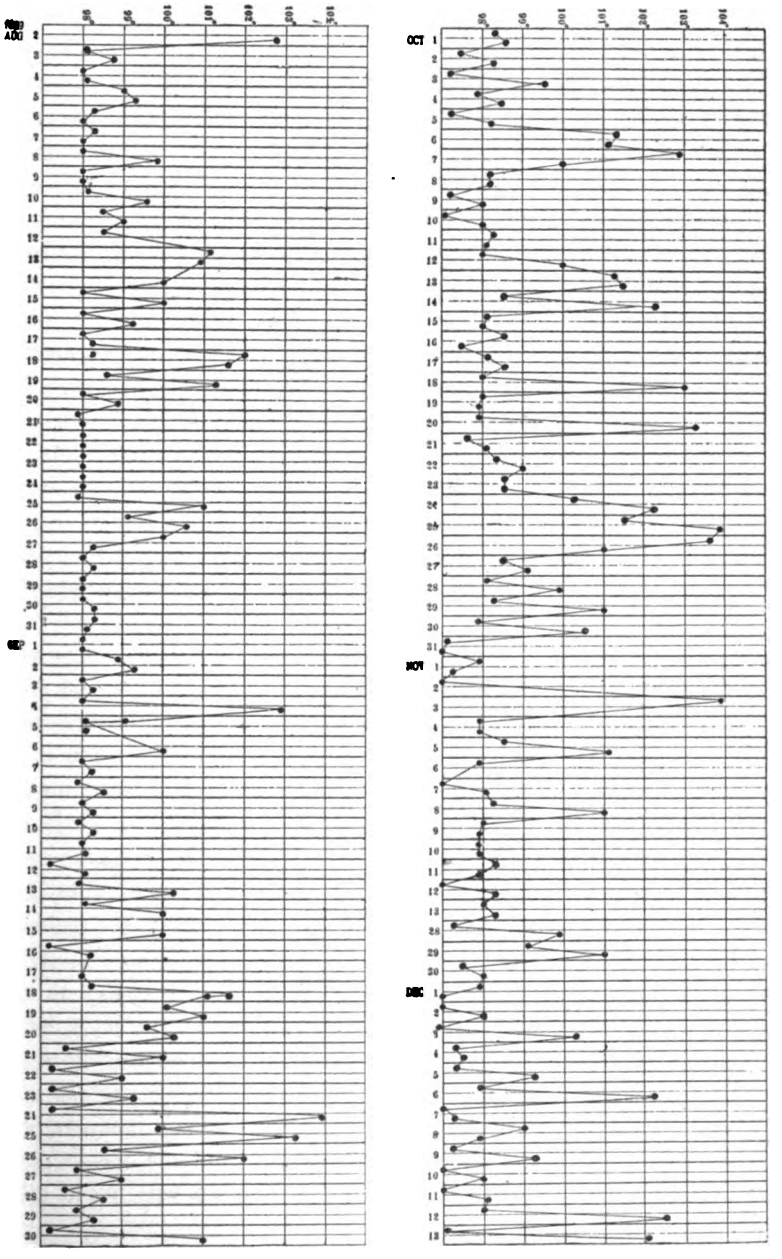

TEMPERATURE RECORD. (*See p. 20.*)

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of the cases, I, III and VII, the jaundice for months together was of the most intense grade.

It is possible that cases of intermittent pyrexia may occur without jaundice, owing to chronic obstruction of a main duct in the liver. I have not met with such a case, but Magnin[1] refers to one under Charcot's care.

Second: *Fever.* This, in well-developed paroxysms, begins with a sharp rigor. I have rarely seen in intermittent fever chills of greater severity. In Case III, in particular, the large, stout woman would, during the rigor, shake the entire room and cause the small, wooden house in which she lived to vibrate. It may be represented, however, only by a sensation of cold, a creeping chill, in contra-distinction to a shaking one. The fever rises suddenly, and, as shown in the chart, may reach from 103° to 105°. At first dry and pungent, the skin gradually becomes moist, and usually within from two to five hours of the commencement of the rigor the patient is bathed in perspiration. The entire duration of the fever is from six to twelve hours; rarely does it persist for an entire day. Defervescence takes place rapidly when the sweating begins. Although the rule is for the paroxysms to present the usual stages, as here described, there were in each of the cases lesser attacks, often of fever alone or of fever with sweating. Slight rises of temperature without chills are indicated in the temperature chart. Sweating was occasionally seen without the fever. In Case III, in particular, local and general sweating was much complained of. The paroxysms occur at irregular intervals, but I have seen them recur daily for a week or ten days. They may present a tertian or a quartan type, and in such cases the diagnosis of ordinary ague may be made. In Case III, the paroxysms recurred for weeks on Friday.

Third: *Pain* of some sort is as a rule present. It may, but certainly does not always, precede the rigor. In some cases it is not at all a striking feature, and the most intense paroxysms may be quite painless or only accompanied by a sense of gastric distress. It may have all the characteristics of genuine hepatic colic, agonizing, griping pain in the liver-region, with the associated symptoms, feeble pulse and clammy skin. In several of the cases the pain was not at all a distressing symptom.

[1] Loc. cit.

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Fourth: *Gastric disturbances.* Vomiting often precedes or accompanies the attacks, and frequently before its onset the patient complains of loss of appetite or nausea; the tongue becomes furred, and it seemed very often as if a gastric catarrh really initiated the paroxysm.

The condition of the patients in the intervals between the attacks is a point of considerable importance. They are often well enough to resume their work, or in the case of women, to do light household duties. There is not progressive deterioration of health and strength, such as we meet with in malignant disease. With the exception of Case I, who had been ill three years, the patients were all well nourished, some of them fat; even Case VII, who had been jaundiced, she said, for ten years, and who to my knowledge had been so for three years, had a very fair layer of panniculus.

Regnard[1] found in one case that the excretion of urea was diminished during the attack. Only in Case III was a careful study of the urea made during the attacks, but no special diminution was found.

Diagnosis.—The significance of hepatic intermittent fever cannot be appreciated without taking into account the associated group of symptoms, and when these are present it points clearly to obstruction of the common duct by calculus. The condition of the bile-passages in these cases is one of catarrhal, not suppurative, cholangitis.

Chronic obstruction of the bile-duct, either by stenosis or by gallstones, may persist for months without inducing this intermittent pyrexia, as illustrated by the following cases:

Gallstones in the common duct. Chronic jaundice. No fever.

A man, aged 77, was admitted to Dr. Curtin's ward in the Philadelphia Hospital suffering with jaundice. He was a weaver by trade and a moderate drinker. He had had jaundice on two previous occasions, and had been in the out-ward for several months, having been jaundiced for nearly a year. Careful inquiry from the attendants, and from the man who occupied the next bed, failed to elicit any history of chills or sweating. When admitted to the hospital he had profuse diarrhœa; the abdomen was distended, and evidently there was fluid in the peritoneum; he was extremely feeble; the stools were grey

[1] Quoted by Charcot, loc. cit.

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and the urine high-colored and contained bile-pigment. He died on the fifth day after his admission.

I made a dissection with Dr. Atlee, and the following conditions were found: Deeply jaundiced; moderate ascites; liver small, surface granular; gall-bladder slightly distended, projecting one inch (2.5 cm.) beyond the liver margin; the common, hepatic and cystic ducts were greatly dilated. When the duodenum was opened, a nodular body projected beneath the mucous membrane above the bile papilla. This could be felt as a hard body within the head of the pancreas, and was at first thought to be a cancerous mass. A probe was passed through into the orifice of the duct, and on squeezing above the pancreas a bile-stained mucus flowed from the orifice. The nodular mass proved to be a gallstone the size of a cherry firmly impacted into the ampulla of Vater. It could neither be pushed into the common duct nor into the duodenum. A second stone the size of an olive was free in the duct, in which it could be moved up and down. The common duct admitted the index finger, and its main branches in the liver admitted the little finger. The gall-bladder was moderately dilated; contained no stones; the cystic duct was free. The bladder and ducts contained a bile-stained mucus. The liver presented the appearance of ordinary cirrhosis. The kidneys were swollen and bile-stained.

That stenosis of the common duct may persist for months, or years, without inducing chills and fever, is illustrated by the following case:

Stenosis of the common duct. Jaundice of fourteen months duration. No fever.

Hannah C., aged 35, admitted to the Montreal General Hospital, September 25th, 1880, with obstructive jaundice of two months duration. The attack had followed diarrhœa, and had come on without any pain. She remained under observation for nearly a year. The skin was of olive-green color; the stools clay-colored; the urine dark greenish-brown; the liver appeared greatly enlarged, the dullness in the middle line extending four inches (10.2 cm.) from the xiphoid cartilage, four and one-half (11.5 cm.) inches from the sixth interspace, and four inches (10.2 cm.) from the seventh interspace in axillary line. She had frequent severe headaches and occasional attacks of pain, associated usually with vomiting.

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The temperature record, which extended over the entire period of her stay, occasionally showed an elevation of two or three degrees, but she never had chills. The liver increased in size, and on May 21st the note was as follows:

"The liver has gradually enlarged until it now fills a large part of the abdomen, extending in the middle line below the navel and in the flank nearly to the crest of the ilium." She died in August, 1881, of gradual asthenia.

Autopsy.—There was moderate emaciation. The liver was enlarged but not so much as was expected, owing to its vertical position. The surface was smooth and of a deep olive-green color. The common bile-duct was pervious to a small probe, but the first inch and one-half from the orifice it was extremely narrow, the wall darkly pigmented and the lining membrane rough. Above this part the duct was greatly dilated and the walls thickened. The gall-bladder was moderately distended; the walls were hypertrophied, and the lining membrane rough and shaggy. It contained three small stones. The hepatic duct and the branches in the liver of the first, second and third dimensions were enormously distended, forming elongated sacculi. The duct passing to the right lobe admitted three fingers with the thumb between them. The lining membrane of the dilated passages was smooth, not ulcerated, not thickened. The dilatation was confined entirely to the branches above named, the terminal branches being little, if at all, affected. There were no dilated ducts to be seen beneath the capsule. The contents of the duct and of the gall bladder consisted of clear mucoid fluid. The tissue of the liver was smooth and the acini well marked. There were no cirrhotic changes.

These instances show that it is not the obstruction alone which induces the intermittent fever; there must be something superadded, probably the ferment-producing agents, the micro-organisms, which, as we shall see, have been found in two cases.

From a practical standpoint suppurative cholangitis is the only affection from which gallstones with hepatic intermittent fever is to be differentiated. The post-mortem examination in cases I and V, and numerous observations which I have found in the literature, show conclusively that the intermittent pyrexia in these long-standing cases is not necessarily associated with suppuration in the ducts. But, unfortunately, suppurative cholangitis is most frequently caused by

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blocking of the common duct with a stone; and it is important to determine in a given case the onset of suppuration. In deciding this, stress may be laid upon the following points:—(1) increased tenderness in the hepatic region with possibly enlargement of the gall-bladder, as this is a more common event in suppurative cholangitis than in simple obstruction of the duct; (2) the more frequent return of the paroxysms, and in some instances the irregularly remittent character of the fever; (3) the jaundice is not so intense in suppurative cholangitis, and we do not see the remarkable deepening in color after the paroxysms; and (4) the general condition of the patient in the intervals is very different in the two conditions. When suppuration exists there are rarely the prolonged periods of apyrexia, the freedom from distress and the general betterment which we see in cases of simple gallstone obstruction.

There may be, however, the greatest difficulty in deciding, and, after all, in the question of treatment it does not make much difference. I recently dissected a specimen of cholangitis brought to me by my friend Dr. Lainé, of Media, Pa., which was removed from a woman, aged 76, who had had from June until September, chills, fever and sweating, recurring at irregular intervals, either daily, or every third, fourth or seventh day. The liver was tender; no tumor could be felt; the symptoms were evidently pyæmic, and there was inflammation of the right parotid gland. The temperature record, a copy of which Dr. Lainé gave me and has kindly allowed me to reproduce (*see p. 27*), may be compared with the one previously given. The chills recurred more frequently, and the temperature is altogether more irregular than in any case of ordinary hepatic intermittent fever which I have seen. The autopsy showed an abscess of the gall bladder with sinuses. The cystic duct was blocked firmly with a calculus, and another the size of a cherry lay loose in the common duct, not interrupting the passage of the bile. There were two septic abscesses in the lower lobe of the left lung, and there was fresh endocarditis of the aortic valves.

In the chronic obstruction which results from the compression of a cancerous mass either in the head of the pancreas or secondary in the lymph glands, there are occasionally rigors, due to catarrhal or suppurative cholangitis, but the sequence of the symptoms would, I think, enable one to decide between this condition and gallstones.

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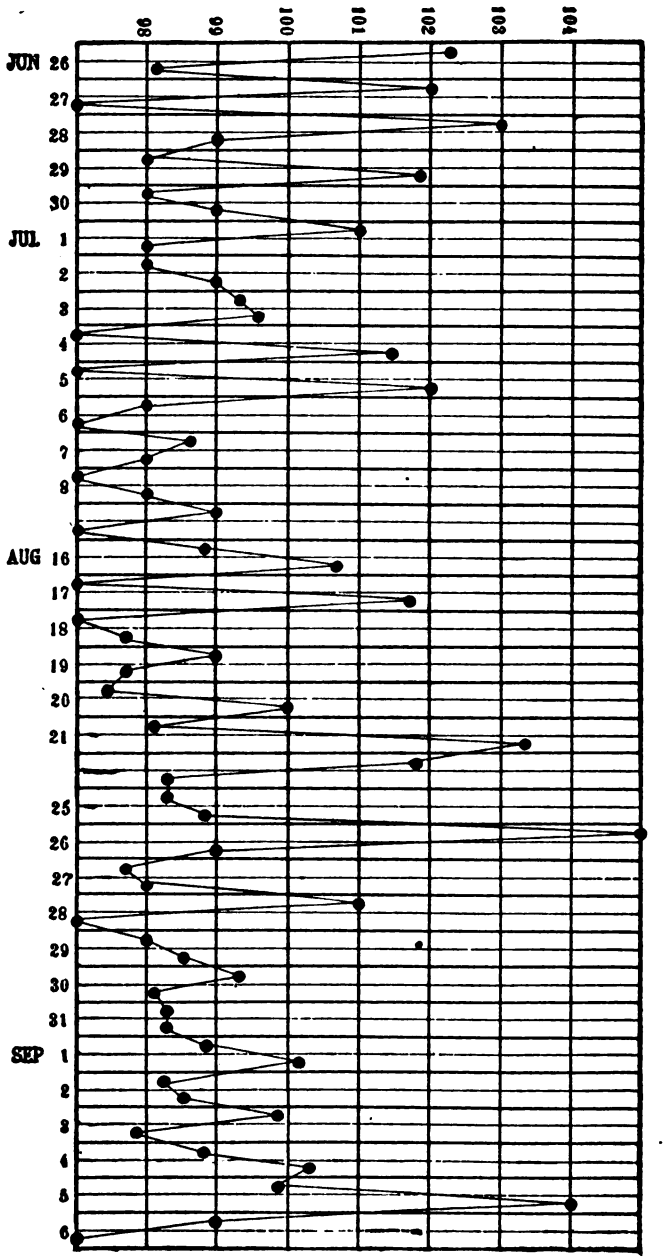

TEMPERATURE RECORD OF DR. LAINÉ'S CASE. (*See p. 26.*)

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The varying intensity of the jaundice and the comparatively easy state of the patient in the intervals between the paroxysms are features which I have not met with, nor seen referred to, in the obstruction by malignant growths.

When the fact is recognized that the lodgment of a gallstone in the common duct may be associated with pyrexia of intermittent type, a confusion of these cases with malaria is not likely to occur. The mistake is, however, very commonly made, and in at least five of the cases here reported the patients were supposed to have chronic paludism, for which they had taken quinine in large doses. The error is a pardonable one when the patient is seen in the interval between two paroxysms, with very slight jaundice and perhaps not more than the lemon-tint of skin seen in chronic malaria. The history of repeated chills is very likely to mislead, and it may require a careful study before the diagnosis can be established. The negative condition of the blood in these cases may be very suggestive, as in cases V and VII, in which the absence of Laveran's organisms led to a revision of the diagnosis.

I have no knowledge of the cases referred to by certain writers, in which a calculus in the duct arouses latent malarial influences, and the paroxysm thus results from the combination of the two factors.

Pathology.—The pathology of hepatic intermittent fever is obscure. Two views have been advanced. Charcot believes that it is due to the production of a ferment in the bile passages, the absorption of which into the blood excites the febrile paroxysms. A certain measure of support is lent to this view by the discovery in the ducts in a case of cholangitis, by Netter and Martha,[1] of a bacillus similar to one of the intestinal organisms.

It is not only in suppurative cholangitis that organisms occur, since in case I, in which the bile-ducts, as stated, contained a yellowish viscid, non-purulent material, Dr. Abbott discovered a short pointed bacillus which did not, in cultures or general characters, appear to correspond with the one described by Netter and Martha.

The occurrence of endocarditis, as noted by these authors, is also extremely suggestive of the action of micro-organisms, and the identity of the organisms in the ducts and those on the heart valves was estab-

[1] Archives de Physiologie, 1886.

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lished by Netter and Martha. Altogether the view of Charcot is one which commends itself most strongly to my mind.

On the other hand, Murchison inclines to the belief that the febrile paroxysms are due to the simple irritation of the stone, not to a septicaemia. To this view, Ord subscribes,[1] stating that the paroxysm of fever is "due to local irritation of the mucous membrane propagated to the central nervous system and resulting in pyrexia, mostly in persons apt to take on febrility, and particularly in persons who have previously had intermittent fever."

It was Budd, I think, who drew the analogy between hepatic and urethral fever, but the analogy to which he referred is rather between the rigor in recent cases of renal and hepatic colic and in the so-called catheter fever. There is, however, a renal intermittent fever, closely analogous to the hepatic form. It may occur, first, in tubercular pyelitis; second, in calculous pyelitis; and third, in rare instances of stone in the pelvis, without chronic suppurative pyelitis. The cases in the last category present a curious analogy to hepatic intermittent fever, due to gallstones, and without suppurative cholangitis. There are intense rigors, the temperature rising to 104° and 105°, with great pain in the renal region and distinct changes in the character of the urine. In a case of the kind which I had an opportunity of studying for several months, the paroxysms recurred at intervals of a few weeks; in each one the urine became somewhat turbid but not purulent. No enlargement of the kidney could be detected, but there was decided sensitiveness in the left renal region. In the intervals of the attacks, the patient was perfectly well and the urine became clear.

In all of these cases the obstruction is not complete, as shown by the presence of bile in the stools for long periods at a time. The association of the chills and fever with intensification of the jaundice must be more than accidental. The two must be correlated in some way, in all probability through a transient impaction of the stone in the duct. Such a condition might induce the chill, either through reflex irritation as held by Murchison, or by preventing the escape from the bile passages of toxic ingredients—ferments produced by the action of micro-organisms—which are absorbed into the blood instead of

[1] Loc. cit.

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escaping freely into the bowel. The impaction is probably overcome by a gradual increase in the *vis a tergo* until the duct is stretched to a point which permits the calculus to fall back into a wider portion. The pressure may reach such a grade that the stone is forced out, as happened in case II, and very likely in the other cases in which recovery followed.

I have emphasized sufficiently the important diagnostic indications afforded by the hepatic intermittent fever, and a careful attention to the group of symptoms presented should enable us to determine whether, in a given case, gallstones alone are present, or whether suppuration has supervened, and the important question remains as to the prognosis and the treatment in these cases.

Prognosis.—I have been fortunate in the cases which I have seen, as three of them recovered; one after a persistence of the symptoms for three years. Judging from the rarity with which recovery is mentioned in the literature, such cases must be deemed exceptional. The great majority of them follow the course which is sketched in the history of cases I and VI, death resulting from exhaustion or cholæmia.

Treatment.—The remarkable success which has recently been obtained by surgeons, indicate clearly the line of treatment which should be followed, and although the results of opening the common duct have not been so favorable as in cholecystotomy, yet they are sufficiently hopeful to warrant the attempt in every case, either to push the stone into the duodenum, to crush or to extract it.

Of medicinal agents I have not found any of the slightest value, either in preventing the onset of the paroxysm or causing the solution or propulsion of the stone. Certain of the cases were drenched with olive oil, and most of them had taken soda salts and mineral waters. Many, perhaps all, of them had taken quinine in large doses, but it is quite ineffectual, either to control or to prevent the paroxysms.

I have dealt thus at length with this special symptom, or rather symptom-group, so characteristic of obstruction of the common duct by gallstones, as I believe a wider recognition of its importance may be the means of saving valuable lives by timely surgical interference.

Conclusions.—1. In cancer and in cirhosis a certain number of cases present fever of moderate grade, but scarcely distinctive enough to be of value in diagnosis.

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2. Chronic obstruction of the common bile-duct is often accompanied by an intermittent pyrexia, associated with a symptom-group of the greatest diagnostic importance.

3. This pyrexia is not usually the result of suppuration, as has been supposed, but occurs with a catarrhal cholangitis.

4. That it arises from the absorption of a ferment, produced in the ducts, is rendered highly probable by the discovery of micro-organisms, both in the catarrhal (Case I) and in the suppurative cholangitis (Netter and Martha).

5. While recovery may follow, even after months (Cases II and VIII), or even years (Case III), a fatal event is only too common.

6. A recognition of the importance of this intermittent pyrexia and its associated symptom-group, as diagnostic of obstruction of the common duct by gallstones, should, in the present condition of hepatic surgery, lead to more frequent operative interference in these cases.

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SOME REMARKS ON ANOMALIES OF THE UVULA, WITH SPECIAL REFERENCE TO DOUBLE UVULA.

By JOHN N. MACKENZIE, M. D.

The uvula varies greatly in appearance in different individuals. Especially is this true in regard to its length. There are several instances on record, for example, in which it was elongated to such an extent, that it protruded from the mouth, interfering, of course, with speech and mastication. It is often found truncated, and not infrequently bifid. In bifid uvula, both halves may hang side by side or may be curved upon themselves like a snail.

The uvula is occasionally congenitally absent. Cases of this kind are found scattered here and there through the ephemeral productions of several centuries back and in the older works on pathological anatomy known now only to the medical scholar. Of these may be especially mentioned the works of Otto and Meckel. The anomaly is especially interesting, because, as was pointed out by Cuvier, in birds and reptiles, the gecko excepted, the soft palate is wanting and that in mammals the uvula is first found in man and the ape, while in the others its absence or rudimentary appearance seems to be offset by a more considerable development of the soft palate. Put beside this the fact that congenital absence of the uvula has been seen in connection with elongation of the velum, and that in one case the latter closed the pharynx like an abnormal skin, and we may possibly trench very closely on the broader question of reversion to atavian forms.

Passing from the discussion of the above-mentioned anomalies, let me briefly direct attention to the subject of double uvula, which, according to the united testimony of the very few observers who have even mentioned the subject, is one of the rarest of embryological curiosities.

I have met with this condition in several forms, which may be classified as follows: —

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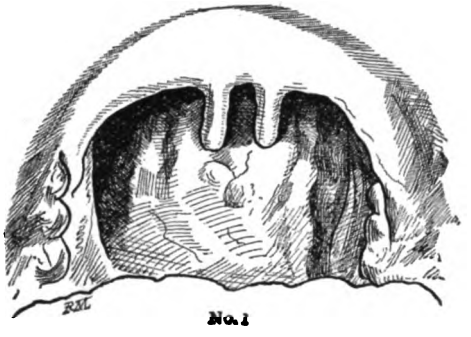

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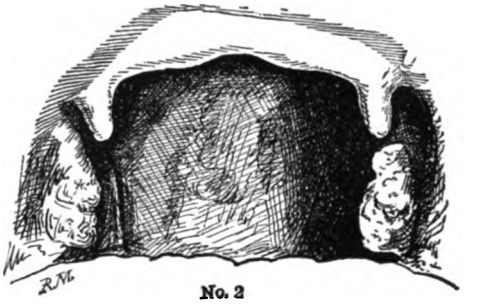

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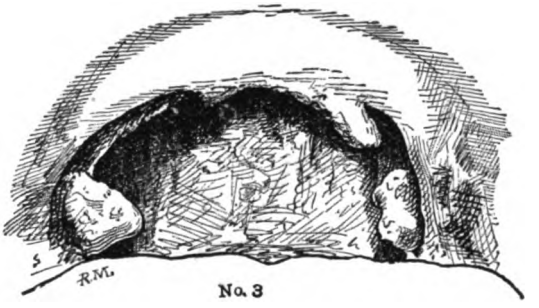

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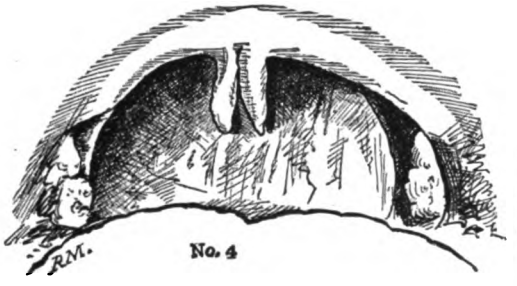

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First variety, in which there are two separate and distinct uvulae hanging apart on either side of the median line of the palate. (Fig. No. 1.)

Second variety, in which there is no central uvula, but two lateral ones, each projecting from the soft palate at, or in the neighborhood of, the junction of the anterior and posterior faucial pillars and directly over the tonsils. (Fig. No. 2.)

Third variety, in which there is one well-formed lateral and externally deflected uvula, its fellow on the opposite side being represented by a mere rudimentary teat, springing from the palate at its junction with the palatine fold. (Fig. No. 3.)

In addition to these varieties, there are two others which represent a sort of transition from bifid to double uvula. They are as follows:—

Fourth variety, in which there are two central uvulae joined only at their insertion into the palate (Fig. No. 4); and finally,

Fifth variety, in which there are two central uvulae joined only at their insertion into a sort of hemispherical bulging downward of the central portion of the soft palate. (Fig. No. 5.)

In my reading I have come across but very few examples of the first variety.

The first is taken from the *Ephemerides of Natural Curiosities* by Meckel,[1] who apparently had never heard of the condition before, and who remarks that it furnishes an excellent illustration of the law, that each half of an organ, when it develops, reaches the same size that the whole organ would have reached, had the embryological process gone on in the natural manner.

The second is a case communicated by P. Tidyman to Professor Chapman,[2] of Philadelphia. The author remarks that he knows of no similar case. The third case is figured and quoted from Mason on *Cleft Palate and Hare lip*, by Dr. Huntington Richards,[3] of New York, in his article on *Cleft Palate* in Wood's *Reference Hand-book of the Medical Sciences.*

The fourth case I take from Lennox Browne.[4] It occurred in a young man, aet. 22. The hard palate was very contracted and highly

[1] Wagner. Dec. III, ann. 1, p. 251. Meckel, Handbuch der path. Anatomie, Bd. I, S. 546.

[2] Phila. Journ. of Med. and Phys. Sciences, Vol. 8, p. 380, 1825.

[3] Vol. II, p. 179.

[4] The Throat and its Diseases, p. 220, London, 1887.

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arched. The abnormality was the source of great discomfort from continued relaxation. Removal was followed by improvement in speech and deglutition.

My own case appeared in the person of a young lady, one of the leaders of Baltimore fashionable society, who consulted me several years ago, concerning a chronic inflammatory condition of the nasal pharynx. Her voice was rather gruff, and she failed somewhat in her high notes.

The second variety I met with, in a young girl from Washington, who came to consult me concerning her voice, which had since infancy been entirely nasal. Receiving a very unfavorable prognosis, of course, she returned home and has never reappeared.

The third variety I have seen in a middle-aged gentleman from Virginia. He came to be operated on for nasal polypi, and the condition of his palate was only an accidental discovery.

The fourth and fifth varieties were accidentally found in two cases of post-nasal catarrh, and seemed to have little effect on the voice.

In addition to the above described anomalies, I would call attention to still another form which I have never seen described. This was the case of a woman who applied at the Johns Hopkins Hospital Dispensary for relief from a retro-nasal catarrh. In the centre of the soft palate was a well-formed truncated uvula, broader above and narrower below, which seemed to be embedded in the palate and covered by its mucous membrane. From the upper border of the base of the uvula on each side commenced the anterior palatine pillar, and from its lower border, the posterior pillar.

The palatine folds, spreading out broadly on either side, from the apparently encapsulated uvula, presented somewhat the appearance, as remarked by Dr. Martinez of Havana, who was present at the examination, of the figures in the text-books, of the uterus with the broad ligaments.

Now, in order to explain these anomalies of the uvula, it will be necessary to turn for a moment to the embryological evolution of the palate and adjacent parts. In the early part of foetal life, the nasal passages and the buccal cavity communicate. Their separation occurs as follows:—About the middle of the second month, after the gums have formed and the dental ridge appears, there pass out from either side two broad plates or processes which meet in the middle line,

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about the middle of the third month, to form the palate. Failure of the processes to unite results in the well-known anomaly, cleft palate. Should the extreme posterior limits of the processes fail to come together, we have the condition known as bifid uvula and double central uvula. But how are we to explain the variety in which the uvulæ are laterally situated in the region of the tonsil and palatine arcade? Why should not the cells which make up the small portion which does not advance to the median line, either disappear or take part in the formation of tonsil or palatine arch? The only explanation which suggests itself is that they are predestined by an inexorable natural law to become uvulæ and nothing else.

Of what clinical or pathological importance are the anomalies described above?

Among the negroes of the South, the palate and uvula have an abiding and far-reaching significance. Under the general term, "the palate is down," they include all diseases of the throat from simple catarrh to tuberculosis and carcinoma; and guided apparently by an intuitive perception of the primal physiological law of reflex action, they adopt as their catholicon in the management of these diseases a kind of treatment which may furnish pabulum for the psychologist. Having tied a tuft of hair, generally on the back of the head, with a red bandana handkerchief or a red tape of some sort, they jerk the lock violently in such a manner as to cause the fallen palate to return to its original place. This superstition is perhaps scarcely less harmful than that which obtains among those of the scientific medical profession, who see in amputation of the uvula the sovereign remedy in the management not only of all pharyngeal and laryngeal affections, but also of all pathological conditions of the nose, and whose rule seems to be—to paraphrase an ancient law of whist—"In case of doubt cut off the uvula."

In the great majority of cases of so-called "elongated uvula," it will be found that the condition is simply that of oedematous relaxation, which is part and parcel of a chronic inflammation of the surrounding parts, and that the uvula will resume its former position with the dissipation of the pharyngeal affection. I emphasize this fact because it brings us to the consideration of one of the chief points of interest which attach to the subject of these remarks, viz: the importance of the uvula in song and speech. That malformations of the

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uvula affect the timbre of the voice goes without saying, but this fact is trivial in comparison with the far more important influence which they may possibly exert in its modification. It has been proven that, in the production of all pure oral sounds, vowels and consonants, the nasal pharynx is completely shut off from the mouth. This is accomplished by an upward and backward movement of the soft palate, which takes place mainly through the instrumentality of the tensor palati muscles. A sort of valve is thus found between the upper and lower pharynx similar to those in the heart and veins (Meyer). Passavant has shown that the palate is aided in this isolation of mouth from nose by a bulging forward of certain fibres of the superior constrictor of the pharynx, upon which the elevated palate rests. As these facts have been demonstrated experimentally by such observers as Czermak, Passavant, Brücke, and others, it becomes at once evident what deleterious influences some of the above described anomalies may have, not only in the process of deglutition, but also in the mechanism of speech and song.

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ON PYRODIN.

By HENRI A. LAFLEUR, M. D.

Of the antipyretics belonging to the anilin group, pyrodin or acetyl-phenyl-hydrazin, has not met with such favor as antifebrin or phenacetin. The observations of Dreschfeld[1] and others prove it to be a most efficient reducer of temperature, but toxic effects were often noticed, due to the action of the drug upon the blood. The statements, however, were made that as the pyrodin first used was impure the bad results were possibly due to the foreign matters; and it was thought worth while to test the action of the chemically pure drug as furnished by Merck.

The following report shows that the drug is a powerful antipyretic, equalling antifebrin in promptness, probably exceeding it in durative action, but that it has a toxic effect on the blood which places it high on the list of dangerous remedies.

CASE I.—*Phthisis.*—Stella P., admitted November 1st. Temperature at 9 p. m., 104.3°; pyrodin, 2½ grains (.16 gms.); 10 p. m., temperature 101°; 11 p. m., temperature 99.5°; 12 p. m., temperature 98.5°; it remained at this point until 2 a. m.; at 3 a. m. it rose to 99°; at 4 a. m. to 100.5°; at 8 a. m. to 102°.

November 2d, 6 p. m., temperature 104°; pyrodin, 2½ grains (.16 gms.); 8 a. m., 100°; 9 p. m., 98.6°; 10 p. m., 97.5°; 11 p. m., 96.5°; 12, midnight, 95.7°; it gradually rose through the morning, and at 4 p. m. had reached normal; at 5 a. m. it reached 101°; for the 24 hours following it did not rise above 102.5°.

November 5th, at 3 p. m., temperature 105°; pyrodin, 2½ grains (.16 gms.); 4 p. m., 104°; 6 p. m., 98°; 8 p. m., 96.7°; it gradually rose, and at midnight was 99.4°, and by 4 a. m. had risen to 103.4°. Temperature for the next 24 hours did not rise above 103.5°.

November 9th, at 4 p. m., temperature 104°; pyrodin, 2½ grains (.16 gms.); 6 p. m., 99°; 8 p. m., 97°; 10 p. m., 97.7°; 12, midnight, 99°. By 2 a. m. it had risen to 102.5°.

[1] Medical Chronicle, 1888.

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November 10th, at 4 a. m., temperature 103.5°; pyrodin, 2 grains (.13 gms.); 5 a. m., temperature 102.4°; 6 a. m., 98°; 8 a. m., 98.2°; it remained about 101° all afternoon; at 8 p. m. had risen to 102.5°. For the next 24 hours after these two doses of pyrodin, which were given at an interval of only 12 hours apart, the temperature remained comparatively low, ranging between 99° and 101°.

She stood the administration well, perspired profusely after each dose, but showed no signs of exhaustion. No special anæmia developed in this case. The blood count did not sink below 1,700,000 per cubic millimetre.

CASE II.—*Pneumonia.*—A. G., admitted October 21st, 8 p. m.; temperature, 104.2°; pyrodin, 2½ grains (.16 gms.); 9 p. m., 103.2°; 10 p. m., 103°; 11 p. m., 102.4°; 12 midnight, 102°; remained at this point until 2 a. m.; it gradually rose until at 5 a. m. it reached 103.8°; pyrodin 2½ grains (.16 gms.); 6 a. m., no effect; 7 a. m., 102.4°; 8 a. m., 102.1°; 9 a. m., 102°. No special influence noticed on the pulse until after the second dose, when the pulse which had ranged from 130 to 150, fell to 100. Very slight perspiration after these doses, and a good deal of restlessness. In this case the pyrodin had practically no influence in reducing the fever.

CASE III.—*Phthisis.*—Zadie C., aged 18, admitted October 2d, ill for one year. Temperature range up to October 4th had been from 101° to 103.5°.

October 4th, 2 p. m.; temperature, 103°; pyrodin, 5 grains (.32 gms.); 3 p. m., temperature, 100°; 4 p. m., 96.4°; 5, 6 and 7 p. m., remained about 96.5°; at 8 p. m. the record was 95°; there was then a gradual rise, reaching the normal about 1 a. m.; at 4 a. m. had risen to 101°; the temperature remained below 102° for a week. On the 11th, at 8 p. m., temperature 103°; pyrodin, 2½ grains (.16 gms.); by 10 o'clock it had fallen to 98°; to 97° by 11 p. m.; and it remained below normal until 8 a. m., and did not reach 102° until 12 midnight.

CASE IV.—*Malaria.*—Minnie S, aged 23, admitted October 11th, ill for seven days. Had a chill on the night of the 4th, none since. She looked very ill, and was thought to have typhoid fever. At 7.15 p. m., temperature 105°; pyrodin, 2 grains (.13 gms.); 8 p. m., 103.5°; 9 p. m., 101°. It slowly dropped through the night, and reached

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98° at about 3 a. m.; it continued to fall throughout the morning, and at 12 m. was 95.2. By midnight it had risen again to 104°.

October 13th, 12 m.; temperature, 104°; pyrodin, 2½ grains (.16 gms.); at 1 p. m., temperature, 100°; 2 p. m. and 3 p. m., 100°; at 4, 5 and 6, a gradual rise to 103.5°.

CASE V.—*Malaria.*—J. P., aged 29, admitted October 4th, and was suspected at first to have typhoid fever. On the 5th, at 4 a. m., temperature, 104.5°; pyrodin, 5 grains (.32 gms.); 5 a. m., temperature, 101.5°; 6 a. m., 100°; 7 a. m., 98.5°; 8 a. m., 98°; up to 4 p. m. the temperature did not rise above 99°; at 11 p. m. he had a slight chill, and the temperature rose to 104°; pyrodin, 2½ grains (.16 gms.); at 12 o'clock temperature was 104°; throughout the morning the temperature fell about a degree an hour; by 6 a. m. it reached 100°, continued to fall through the morning, and at 2 in the afternoon was 97°.

October 7th, 1 a. m.; temperature, 104°; pyrodin, 2½ grains (.16 gms.); temperature fell gradually, and by 6 a. m. was only 101°; by 8 a. m. 99°.

He sweated after each dose, but there was no special constitutional disturbance and no depression of the circulation.

CASE VI.—*Malaria.*—Charles S, aged 28, malaria of irregular type, lasting ten days; admitted October 31st. On November 2d, without any chill, temperature rose to 103.8°; pyrodin, 2½ grains (.16 gms.); temperature fell gradually, and by 8 p. m. was 100°; by 10 p. m., 97.5°.

The following day the temperature was nearly 104°; no pyrodin was given, and the temperature fell throughout the afternoon and night to 97° by 4 a. m.

On the 4th, temperature at 3 p. m. was 104°; pyrodin, 2 grains (.13 gms.); by 6.30 temperature had fallen to 98.2°.

CASE VII.—*Malaria.*—John F., aged 30, admitted September 21st, with malaria of more than a month's duration. On the 21st temperature rose to 107° after a chill. Between 6 p. m. and 2 a. m. it fell exactly 10 degrees, dropping in 8 hours from 107° to 97°.

On the 23d the temperature was 105° at 8 p. m., and dropped to 98° by 6 a. m.

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On the 24th, at 8.30, it was 105.5°; pyrodin, 5 grains (.32 gms.); temperature fell rapidly, and by 12 o'clock was normal. It remained below normal until 6 p. m. the following day, when he had another chill.

The pyrodin in three hours and a half brought the temperature to normal, whereas, judging from the former paroxysms, it would have taken from eight to ten hours in the normal defervesence.

CASE VIII.—*Malaria.*—N. M., aged 28, ill for two weeks. At 1.30 p. m. temperature was 105°; pyrodin, 5 grains (.32 gms.); by 6.30 temperature had reached 100°; gradually fell, and by 10 p. m. was 97°.

Perspired profusely after the dose, but no depression.

CASE IX.—*Typhoid Fever.*—J. R., aged 32, admitted about the end of the second week, having come from Hamburg on a steamer. For the first five days in the hospital, the temperature ranged from 101° to 103°. On the 7th, at 4.30, temperature was 104°; pyrodin, 2½ grains (.16 gms.); 5.30 p. m., 103°; 6 p. m., 102°; 7 p. m., 99°. From this point it gradually rose, and at 12 reached 102.5°. There was no special depression.

CASE X.—*Typhoid Fever.*—Zed. S., aged 38, admitted with typhoid fever on the eighth day of disease. The temperature range had been from 101° to 104°, and the high fever did not seem much affected by sponging. October 3d, at 11.30 p. m., temperature 104°; pyrodin, 5 grains (.32 gms.); 12.30, temperature 100°; 1.30, temperature 98°; it remained about normal throughout the next day, not rising above 98° until 6 p. m.; throughout that night it was about 99°; he seemed very comfortable, and was not at all depressed by the dose. Practically for more than 24 hours the temperature was kept down by this single dose.

On the 5th, by 11 p. m., temperature had risen to 103°; pyrodin, 2½ grains (.16 gms.); by 1 a. m. it had fallen to 98°; it remained below 100° until 6 a. m.

On the 7th, at 4 p. m., it was 104°; pyrodin, 2½ grains (.16 gms.); temperature at 5 p. m., 99°; remained below 100° until 2 a. m.

9th, 5 p. m., temperature 104°; pyrodin, 2½ grains (.16 gms.); 6 p. m., 102.5°; 7 p. m., 101.5°; 8 p. m., 100°; it gradually rose through the morning, but did not rise above 101°.

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10th, 12 m., temperature 103.5°; pyrodin, 2½ grains (.16 gms.); reduction by 2 p. m. to 100.5°; 3 p. m., the same; a gradual rise towards evening, reaching by midnight 103°.

On the 11th, at 6 p. m., temperature 104°; pyrodin, 2½ grains (.16 gms.). By 8 p. m. a gradual fall to 100°, followed by a rise to 102° by 2 a. m.

CASE XI.—John K., aged 30, admitted November 10th. At 1 a. m., on the eleventh, temperature 103°; pyrodin, 2½ grains (.16 gms.). Temperature dropped slowly after the first hour; was 100° at 2 a. m., rose again by 4 a. m. to 102°, and fell by 7 a. m. to 100°. He perspired moderately; had well marked delirium after the dose. Called himself a lunatic, and said he felt as if he was becoming insane.

CASE XII.—*Typhoid Fever.*—Harry H., admitted October 15th, about the twelfth day of the disease. Temperature ranged from 102° to 104°, from the 15th to the 18th.

On the 18th, at 10 p. m., temperature 103°; pyrodin, 2½ grains (.16 gms.); 11 p. m., temperature 102.5°; 12, midnight, 99°; 1 a. m., 97°; 2 a. m., 97°; 3 a. m., 101°. From this hour, until 12 the next day, the temperature did not rise above 101°. The dose was not followed by any unpleasant effects, and there was not much sweating.

In the preceding cases twenty-seven doses of pyrodin were given, usually two and a half grains (.16 gms.), and in almost every instance there was a marked antipyretic effect, quite equal to that of antifebrin. There was not more depression than is seen after fifteen grains (.96 gms.) of antipyrin or five grains (.32 gms.) of antifebrin, nor was the sweating excessive. In Case XI there were transient mental symptoms.

The following cases illustrate the untoward effects of the drug in inducing acute anæmia by destruction of the blood corpuscles.

CASE XIII.—*Phthisis, Acute Anaemia and Leukaemia.*—A. G., aged 34, admitted October 24th, 1889, complaining of cough, shortness of breath and loss of flesh. Family history good, no tuberculosis; patient had been well and strong most of his life, with the exception of small-pox in 1883. The present is his first illness since childhood. In December of last year he caught a heavy cold—was not laid up

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in bed, but had a cough throughout the winter, and was at times short of breath. In the spring he began to lose flesh and strength rapidly; noticed afternoon fever, and began to sweat at night; his digestion also became disturbed, and in July he stopped work.

On admission, the following note was made: emaciated, feeble, temperature 102°; chest long, narrow; expansion slight, more on the left than on the right side; clavicular spaces marked; resonance defective on right side to third rib; signs of large cavity at right apex and of a smaller one at left apex behind; the pulse was 110; tubercular bacilli and elastic tissue found in the sputa; spleen enlarged, palpable; the blood showed no special change. From the 24th to the 28th, the temperature range, in hourly and two hourly observations, showed a continued elevation reaching to 103°. Pyrodin was ordered in two grain doses (.13 gms.). Beginning at 12 noon, on the 30th, he took up to 6 p. m., on the 31st, ten grains (.64 gms.), with the effect of keeping the temperature continually below 98°. He seemed much better and slept well. It was continued at intervals through November 1st, 2d and 3d, so that up to 6 a. m., on the morning of the 4th, he had taken twenty grains (1.28 gms.) in six days. The temperature during this time remained between 98° and 100°. He seemed comfortable, the sweat was occasionally profuse after the pyrodin, but there was no extreme depression. On the 4th it was noticed that the skin had a uniformly lemon-yellow tint, and that the conjunctivae were a little yellow. He had become distinctly anaemic on the 4th and 5th; the urine was also of a decidedly cherry-red color; on the 6th the following note was made: "he has become much more anaemic; the whole skin has a light yellow tint, similar to that seen in pernicious anaemia. The contrast in his appearance now and ten days ago is most striking. The spleen is not more enlarged than it was; the blood count was as follows: red corpuscles 1,775,000 per cubic millimetre, haemoglobin under 20 per cent. Proportion of white to red 1:29. The red corpuscles present moderate variations in size and shape. There was a number of microcytes. The leucocytes were chiefly of the multinuclear form; staining by Ehrlich's method shows the presence of a number of nucleated red corpuscles."

On the 9th the blood count was 1,031,000; proportion of white to red corpuscles, 1:30. No retinal changes.

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Urine was cherry red in color, alkaline in reaction; specific gravity 1,015; no albumen, no bile pigment. He sank rapidly on the 10th, and died at 3 p. m. The temperature, from the 4th, when the pyrodin was omitted, gradually rose and reached between 102° and 103°.

The post-mortem examination showed extensive tubercular disease, with cavities in both lungs, most marked in right apex. The heart was normal; the spleen was enlarged to about three times its normal size, was tolerably firm and not amyloid. The liver was slightly enlarged, anæmic and fatty; the kidneys were anæmic.

CASE XIV.—*Hysteria.*—A. B., aged about 35, subject of profound hysteria, with attacks of severe superficial pains, for which she had repeatedly taken antipyrin and antifebrin without much effect. She was a well nourished, moderately stout woman, with a fair color. On November 2d, 6th, 7th and 8th, she was given 2½ grains (.16 gms.) of pyrodin, taking in all 10 grains. It seemed to have no influence whatever upon the pains; she was not prostrated after the doses, nor did she sweat very heavily. On the 12th it was noticed that she had a distinctly sallow tint; this condition increased until by the 18th there was a well pronounced anæmia, the blood count showing red corpuscles 2,114,560; ratio of white to red, 1:169. Under iron in full doses the blood condition improved and the sallow tint of the skin disappeared.

CASE XV.—*Typhoid Fever.*—Sophie E, aged 19, admitted November 1st, on the eighth day of the disease. November 1st, 9 p. m., temperature 104.2°; 2½ grains (.16 gms.) pyrodin; 10 p. m., temperature 101.2°; temperature fell gradually, and at 1 p. m. was 97.4°; 2 a. m., the same; it gradually rose, and at 4 a. m. was 100.3°; and at 6 a. m. was 103.2°.

November 2d, 10 p. m., temperature 103°; pyrodin, 2½ grains (.16 gms.); by 12 o'clock temperature was 99°; no further reduction. From 1 a. m. to 6 a. m. temperature remained about 100°.

November 4th, 5 p. m., temperature 104°; 2½ grains (.16 gms.) pyrodin; 6 p. m., temperature 100°; 7 p. m., temperature 98°; it remained at this point until 11, when it began to rise, and at 1 a. m. had reached 103°. She sweated profusely.

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November 6th, 2 p. m., temperature 104°; pyrodin, 2½ grains (.16 gms.); 3 p. m., temperature 103°; 4 p. m., 101°; by 5 p. m. it had risen to 103.5°; at 6 it was 102°, and at 8 it was 102.3°.

She took in all 10 grains (.64 gms.) of pyrodin between 9 p. m. on the 1st and 3 p. m. on the 6th. About the 9th it was noticed that she was becoming very sallow with a light lemon tint. Within a few days the pallor became quite intense. Blood examination showed on the 15th, 1,556,000 red corpuscles; proportion of white to red, 1:166. Urine had somewhat a cherry red color.

In spite of the persistence of high fever and development of an acute pneumonia, the pallor distinctly diminished. The blood count, however, on the 25th did not show much change; a little over 1,500,000 red, per cmm. Ratio of white to red, 1:120. She made a satisfactory convalescence.

The highly poisonous nature of the drug and its mode of action are well shown by the following experiments:

I. Male rabbit; weight 44½ oz. (1260 gms.). November 20th–November 22nd was given four 1-grain pills (.065 gms.) of pyrodin; died during night of November 22nd.

Autopsy.—General anæmia; blood pale and watery; gives haemoglobin spectrum. Fatty degeneration of heart muscle, not extensive. Spleen enlarged; dark and hyperaemic. Kidneys: capsule easily detached, surface dark brown, with a few dark red patches, cortex dark brown, contrasting markedly with pale greyish medullary portion. Bladder contains about 25 cc. of dark red turbid urine, with some soft reddish clot.

Microscopic Examination.—Kidneys: In the collecting tubules especially, but also in the convoluted tubules, there are dark reddish-brown casts, which appear to consist entirely of granular pigment. No blood casts. Renal epithelium not sensibly altered. Urine: Casts similar to those seen in section of the kidney, with much pigmentary *débris*. No blood corpuscles.

II. Rabbit; weight about 45 oz. (1275 gms.). November 25th–30th was given 10 ½-grain pills (.032 gms.) of pyrodin; died during night of November 30th.

Autopsy.—Intense anaemia of all parts of the body except the kidneys and spleen. Blood pale and watery. Right heart filled

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with fibrinous clot. Heart muscle pale and easily torn. Spleen enlarged; dark and succulent. Kidneys: appearance similar to those of rabbit No. 1, but brown color is not so intense, and the reddish haemorrhagic-looking patches are not present. Submucosa of stomach presents numerous small ecchymoses of a dark brownish-red color. Bladder: Contains 30 cc. of turbid brownish-red urine. Mucous membrane normal.

Microscopic Examination.—Heart muscle shows very marked and uniform fatty degeneration. Kidneys: dark brownish-red casts of granular pigment in some of the collecting and convoluted tubules, but not so numerous as in rabbit No. 1. Some of the tubules are filled with epithelial *débris*. Urine: brown-red casts fewer in number. There are in addition well marked hyaline casts, with epithelial cells adherent to them; no red blood corpuscles. Urine and blood give haemoglobin spectrum.

These clinical and experimental results correspond with those of Guttmann and to the more recently published account from von Ziemssen's clinic, by Ziegler.[1]

While as prompt and as efficient a reducer of fever as the other members of the anilin group, pyrodin thus possesses in a more marked degree their poisonous properties, and should not be given in doses exceeding two grains (.13 gms.), or continued for longer periods than two or three days.

[1] Deutsches Archiv. für klin. Medicin, Bd. 45, Heft 3 and 4 (Oct. 24, 1889).

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CASES OF POST-FEBRILE INSANITY.

By WILLIAM OSLER, M. D.

One of the most distressing accidents which can follow an acute disease is the development of mental symptoms, which may take the form of excitement, depression, loss of mental power, delusions, or hallucinations. In medical practice these cases form the counterpart of the insanity seen by surgeons after operations, and of the puerperal insanity described by obstetricians. It is a somewhat rare condition, and in a tolerably large hospital experience, I had seen, to January, 1888, but two instances: one after pneumonia, and one after typhoid fever.

At the November meeting of the College of Physicians, of Philadelphia, Dr. H. C. Wood[1] discussed the relation of these forms to each other, and proposed to consider them as instances of "Confusional Insanity," with one common fundamental brain condition, viz: impaired nutrition with consequent exhaustion of the nerve centres. Of the cases which he reports, three followed operations and one typhoid fever. The subject of insanity in surgical practice has recently been brought to the notice of the profession by Shepherd,[2] of Montreal, and by T. Gaillard Thomas,[3] of New York. A report of the following post-febrile cases, five in number, which have been under observation within the past eighteen months may be of interest, as they illustrate the important points in the clinical history of this condition.

CASE I.—*Pneumonia. Slow convalescence with development of hallucinations and delusions.*

A. B., aged 42, farmer, applied at the Johns Hopkins Hospital June 20th, 1889. Family history is good; no insanity or any nervous trouble. Patient has been a healthy man and a hard worker; was a staff officer in the war. In March he had a severe attack of pneumonia, the convalescence from which was slow, but at the end of

[1] University Medical Magazine, December, 1889.
[2] American Journal of the Medical Sciences, December, 1888.
[3] Medical News, 1889.

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six weeks he was able to be up and about. The mind at this time was perfectly clear, and had been so throughout the illness. About two weeks after convalescence he was noticed to be a little odd and peculiar; was low-spirited and depressed, and began to imagine all sorts of troubles; thought that he was in serious financial difficulties. He was never violent, simply melancholic.

When seen the patient was pale, with a sad, depressed expression of face. He would respond to questions, but not promptly, and speech seemed slow and hesitating. It was only with difficulty that any account could be obtained from him, of his feelings. His chief worry seems to be that he has lost the respect of his friends, and that people are plotting against him. His friends were advised to keep him at home carefully guarded.

September 21st. Patient seen to-day by Dr. Toulmin. He is looking, and has been, much better, has gained in weight, complexion is good and expression is cheerful. He still has hallucinations, and thinks that he has done something which he should not. Twice he has been violent, but was restrained without much difficulty. He seems to be progressively improving.

CASE II.—*Typhoid fever; severe attack with much delirium. Mania during convalescence. Gradual recovery after four months.*

Mary J., aged 28, seen with Dr. Fussell on February 19th, 1888. Family history bad; a sister died of phthisis. No mental troubles.

The patient in January had an attack of mild typhoid fever, in which the mind was clear, the pulse not above 100, temperature not above 103°; the rose spots were well marked.

On January 29th, with the temperature 101°, the pulse 120, she was delirious for the first time. On the 30th and 31st she was constantly talking, chiefly on religious subjects. She tried to get out of bed, and was full of delusions. Throughout the first week of February, her temperature was not above 101°, but the condition of delirium was most intense, at times becoming quite maniacal.

I saw her on February 16th, in the following condition:

The temperature had been normal for at least a week. She was emaciated, and had a wild, anxious expression. She sat up in bed, and could not be induced to lie down. She talked incessantly, chiefly upon religious subjects. It was with difficulty that she could be kept

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in bed. She had taken a violent aversion to her husband. She had both hallucinations and delusions. Unless under the influence of hyoscin she rarely slept. It was found necessary to remove her to the Pennsylvania Hospital for the Insane on February 22d, where she remained three months, when she returned home sound in mind but very weak.

In September she died of what was stated to be ulceration of the bowels.

CASE III.—*Typhoid fever of moderate severity. During convalescence development of delusions. Recovery after six weeks.*

Richard F., aged 30, seen with Dr. Gibb, November 20th, 1888. He had been ill since about the fourth of October, with a perfectly well characterized typhoid fever. The temperature was never very high, but the fever persisted for between three and four weeks. During the fever he had delirium, which set in early in the first week; it was, however, of a quiet character. When I saw him, he had had no fever for nearly two weeks. He was pale, but not much emaciated. The pulse was under 100, and the temperature normal. The tongue was a little furred. He was subject to distinct delusions. He did not know where he was; did not always recognize his wife, and constantly talked about events which had never happened. He was never maniacal. Dr. Gibb thinks that the delusions of the febrile state and of his present condition presented no distinct break. The symptoms persisted for a month subsequent to my visit. Convalescence was very slow, but with the recovery of his strength, the delirium and mental weakness disappeared.

CASE IV.—*Typhoid fever, mild attack. Gradual development of delusions. Slow, halting speech. Recovery.*

Henry C. P., aged 14, applied at my clinic, at the Infirmary for Nervous Disease, on March 6th.

As a child he was always healthy. He is of fair intelligence, and has made good progress in his studies. There are no nervous disorders in the family.

On December 22, 1888, he was taken ill with typhoid fever, apparently a mild attack. Temperature never rose above 103°. From the outset the head symptoms were well marked. He cried and

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whined a great deal. He did not know his mother; he had a delusion that she was dead. The fever only lasted for two weeks; the rash was well marked and there was diarrhœa. The mental symptoms persisted for nearly four weeks after the temperature had fallen to normal.

In the early part of February he seemed to have recovered—at any rate to have gotten rid of his delusions; but his parents sought advice as he was, they said, a little queer.

Patient is a bright, well-nourished lad, answers questions rationally remembers all about his illness, and in conversation nothing peculiar would be noticed except that he hesitates and is slow in his speech. His mother says he is very restless, never remaining quiet for more than a few minutes. He is most anxious minded, and constantly thinks something is going to happen to his parents. He frequently wakens at night and behaves strangely. He does not seem to know his father. His slow, halting speech is quite marked, and is a feature which has developed since his illness.

Physical examination, negative. Heart normal. He eats well, and has gained rapidly in weight and in strength. A favorable prognosis was given, and I heard in May that he had recovered.

CASE V.—*Typhoid fever, severe attack. During convalescence development of delusions. Persistence of mental symptoms for ten weeks. Recovery.*

Thomas D., aged 39, mechanic, admitted to the Philadelphia Hospital December 29th, 1888.

He had been off work for nearly six weeks, and for nearly a month of this time had been drinking heavily. For two weeks prior to his admission he had had fever and had been in bed a great part of the time, during which he continued to take much alcohol.

On admission he was rational. Temperature 101.6°, pulse 80. The only noticeable feature was the excessive tremor, which was attributed to alcohol, as his general condition was good. During the first week in hospital his temperature remained about 102°. There were distinct rose spots. He had rambling delusions at night, and would constantly attempt to get out of bed. On the 6th and 7th the temperature registered very low, 96.8°, but did not remain depressed for many hours. There was marked tympanitis on the 9th and 10th.

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From the 12th to the 20th he improved rapidly. The temperature fell to normal, and he seemed to be convalescing rapidly. From this time on he showed marked mental disturbances. He did not recognize his mother, and constantly attempted to get out of bed. He became suspicious and presented many delusions.

Through February this condition persisted. He was very disturbed at night. He was very feeble and looked depressed. Hyoscin, morphia, paraldehyde, bromide of potassium, and chloral hydrate, were all tried in turn without satisfactory results. At one time he was quite violent, and had to be watched day and night. His delusions were chiefly of an unpleasant character. He thought that the persons in the ward were trying to kill him, and that he was being treated badly, and being kept away from his friends.

During the first and second weeks of March, his condition improved; he took food better and seemed to gain in weight. He was more readily interested, and at times seemed quite rational. The recovery was slow but progressive, and I saw him in June, when he seemed quite well.

Two points of practical interest may be mentioned. The prognosis in these cases is usually good. Of the seven cases in all, which I have seen, five after typhoid fever and two after pneumonia, six have recovered, and case I, reported in this paper, will, in time, probably get well. This should be remembered in considering the treatment of these post-febrile insanities, and renders it advisable, if at all possible, to care for the patient, at home. When actively maniacal, as in case II, this may not be practicable, but in general practice among the better classes, with a hopeful outlook and the prospects of recovery within a period of three months, home treatment should at first be tried. Seclusion, incessant watchfulness, absolute rest in bed, with massage and careful feeding constitute the essentials in treatment. It is interesting to note, as in case III, how, with the recovery of strength and improvement in general nutrition, the mind becomes stronger.

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ACUTE TUBERCULOSIS IN AN INFANT OF FOUR MONTHS.

By HARRY TOULMIN, M. D.

Although the recent statistics of Landouzy,[1] Querat,[2] Lannelongue,[3] Leroux[4] and others show that tuberculosis in infants is by no means uncommon, the following case presents features of sufficient interest to make it worthy of record.

Bertha H. M., aged 4 months, white, was admitted to the Hospital, July 20th, 1889, with cough, fever and shortness of breath. She was the third of three children. One sister, five years old, was healthy and strong. The other child died when seven days old, of tetanus. The mother and father were perfectly well. There was no tubercular history in the family, on either side; grandparents healthy. Neither uncles, aunts nor cousins had any lung trouble. Until the child was nine weeks old, they had lived with friends in Washington, none of whom had tubercular trouble. They then moved to their present home, and one week later (six weeks previous to admission) the child was first noticed to be unwell. This house had been vacated three weeks previously by a woman with extensive tubercular lesions of both lungs,—a dispensary patient,—in whose expectoration tubercle bacilli had been repeatedly found.

The child was first seen as an out-patient on the 11th, when the note made by Dr. Osler, was as follows:—"Well nourished, healthy looking infant, who, the mother says, has been ill for six weeks with cough and fever. The chest is well formed; respiration hurried; marked retraction of the lower zone of the thorax; expansion about equal at apices. Percussion shows marked impairment on the right side in front, as low as the fourth rib. The contrast between the two sides is striking. When she cries, tactile fremitus is here much exag-

[1] Congres pour l'Etude de Tuberculose, 1re Session, 1888, Paris.
[2] Paris Thesis, 1886.
[3] Etudes sur la Tuberculosis, Verneuil, 1re fasciculus, Paris, 1887.
[4] *Ibid.*, 1888.

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gerated. The breath sounds are harsh, bronchial, but not typically tubular; and at the end of inspiration there are innumerable crepitant râles. Posteriorly there is impairment of resonance, with râles, in the region of the right scapula. In the left lung in front, the breath sounds are harsh, but no râles are heard. Posteriorly there are fine crepitant râles in the central part of the lung."

The child was seen again on the 15th, when the dullness did not appear to be so marked. The blowing breathing at the right apex was intense, and the râles were numerous and large.

When admitted, five days later, the color was a little dusky; pulse very rapid, too feeble to be counted; temperature 99.8°. On the following morning, the 21st, the color had improved. On examination, there was still defective resonance at right apex; both bases clear. On auscultation, at right apex, anteriorly and posteriorly, the breath sounds were intensely articulate and harsh. Numerous large loud râles were heard. The left apex and base seemed clear. In the evening the pulse was counted at 148. Respiration 44; temperature 101°. Expiration was grunting. Condition otherwise remained unchanged.

On the 22d, after a restless night, but little difference noted. Temperature was 101.8° at 8 a. m., and 100.8° at 7 p. m. Cough was distressing, and color became worse during the day. At midnight the temperature had again risen to 101.8°. At half-past one, on the morning of the 23d, she had a convulsion. Cyanosis was more marked at this time; respiration more frequent; pulse could not be counted; temperature 104°. Death at 6.30 a. m.

Post-mortem, nine hours after death.—Much discoloration; no rigor mortis; superficial fat abundant.

Pericardium smooth, no effusion. Diaphragm corresponds to sixth interspace on both sides.

Thorax. Thymus gland large and normal. Anterior mediastinal glands are large as marbles and cheesy.

Pleura smooth, no adhesions on either side.

Heart large. The auricles and the right ventricle contain blood clots. The foramen ovale is completely closed. The ductus arteriosus is closed at the aortic end, but at the pulmonary artery there is a small orifice, into which a fine glass probe can be passed. There is a small tubercle on the endocardium of the anterior wall of the right ventricle.

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Lungs. The upper lobe of the right lung is scarcely anywhere crepitant, except at the extreme anterior edge. The middle and lower lobes are heavy and slightly crepitant. The visceral pleura is beset with tubercles, most of which have grown into it from the lung tissue. On section the right upper lobe is thickly studded with caseous masses from 5–12 mm. in diameter. The intervening tissue is of deep red color. The middle and lower lobes are stuffed with tubercles, smaller in size, but many of them cheesy. In the right lung towards the diaphragmatic surface of the lower lobe, there is a recent cavity the size of a marble. The left lung is more crepitant and uniformly studded with tubercles of all sizes, some as large as peas. The trachea and bronchi are filled with a tenacious mucus. Above the bifurcation and in the main branches there are tubercles in great numbers. The bronchial glands are swollen, forming a large mass. They are caseous, and on section there is a tubercular abscess the size of a walnut.

The spleen is large and of a purple red color; the capsule is studded with tubercles. On section there are innumerable tubercles throughout the pulp.

Stomach. The mucous membrane presents two small follicular ulcers.

The liver is large and the capsule is covered with small gray tubercles. The substance contains numerous minute miliary granules of such translucency that they can scarcely be seen.

The kidneys show prominent lobulations and contain many tubercles. The bladder and pancreas are normal.

Intestines. The jejunum presents numerous small, deep, follicular ulcers; the mucosa about them is congested and swollen; towards the ileum they become more numerous; many of the Peyer's glands are swollen and present ulcers. The caecum and colon present tubercles and several small ulcers.

The mesenteric and reto-peritoneal glands are uniformly large, and present cheesy masses at their periphery.

The microscopical examination showed tubercle bacilli in the pus of the small abscess in the lower lobe, and they also existed in the stained sections of the organ.

The points of interest in this case may be thus summarized.

It illustrates the notorious difficulty which exists in the diagnosis between simple and tuberculous pneumonic processes in children.

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The area of consolidation in the upper lobe was extensive enough to produce dullness and blowing breathing, while the scattered patches in the rest of the lung produced only harsh breath sounds, and behind, fine râles. It does not seem possible that a diagnosis of tuberculosis could have been made from broncho-pneumonia, or indeed from a lobar-pneumonia, involving the upper lobe. A tuberculous family history would have been suggestive. As usual in infants, sputum could not be obtained, though one may sometimes in the vomit find sufficient expectoration to make a satisfactory examination. Landouzy[1] calls attention to the frequent enlargement of the spleen in the tuberculosis of infants—a condition found, post-mortem, in this case. In the more chronic cases there is of course less difficulty in diagnosis, but even here it may be impossible to obtain positive evidence of the tuberculous nature of the trouble.

Another point of great interest in this case is the source of infection. Since the demonstration of the existence of a congenital tuberculosis—it is true in extremely rare cases—some writers have been inclined to regard these instances of the early outbreak of the disease as pointing to the transmission of the virus in utero, but in this case the parents were perfectly healthy, and there was no evidence of any tuberculosis in the family. The child was breast-fed and had not been given cow's milk. There is, however, a possible source of infection, which must not be overlooked. These people had lived with friends in Washington until the child was nine weeks old, when they moved to their present home, a house vacated three weeks before by a woman with extensive tuberculous disease in both lungs, a dispensary patient in whose expectoration we discovered repeatedly tubercle bacilli. It is quite possible that portions of dried expectoration remaining in the house—and we know from Cornet's investigations that the virus connected with them does persist in the rooms occupied by consumptives—may have proved the source of infection to the child.

It may be worth while to refer to one or two facts in the recent literature indicating that tuberculosis in sucklings is by no means a rare disease. West, in his work on Diseases of Children, gives a table of 497 cases up to the 15th year, among which there were 46 cases under one year and 72 cases between the 1st and 2d year. These

[1] Loc. cit.

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older figures of West do not seem to have attracted the attention which they deserve. Much study has been given to the subject during the past few years by French writers. Querat[1] reports 11 cases in children under 2 years. He quotes also Hervieux, who in 996 autopsies on infants, found 18 under 2 years of age and 10 under 1 year. It is worth noting that in 2 of his cases the disease was found in infants dying before the 15th day. Leroux[2] has fortunately been able to analyze the statistics of the late Prof. Parrot, embracing 219 cases in children under 2 years. Of these there were from 1 day to 3 months, 23 cases; from 3 to 6 months, 35; from 6 to 12 months, 53; a total of 111 under 1 year—from 1 year to 2 years, 108. Of the cases dying under 3 months, 1 was at the 18th day, 1 at the 4th week, 1 at the 5th, 2 at the 6th, 5 at 2 months, 8 at 2½ months, 5 at 2 months and 3 weeks. Pulmonary cavities existed in fifty-seven of these cases, and in only fifty cases was the pulmonary lesion the sole manifestation. This author concludes that we must revise the opinion heretofore held as to the rarity of tuberculosis in early infancy.

Still, in any large group of statistics giving the causes of death in sucklings, tuberculosis does not appear to rank very high as shown by the figures of Frobelius,[3] who states that in the St. Petersburg Foundling Asylum, in the ten years ending 1884, there were 16,581 autopsies on sucklings, only 416 of whom were found to be tuberculous. The probability is that with our present method of anatomical diagnosis this percentage would be greatly increased.

Querat refers to the difficulty which sometimes exists, even post-mortem, of determining whether the broncho-pneumonia is simple or tuberculous. The small grey tubercles may be concealed in islands of pneumonia. Cornil and Babes[4] report just such a case, in which the diagnosis of broncho-pneumonia was made during life, and to the naked-eye inspection tubercles were not evident, but microscopically the process was shown to be tuberculous. Among recent French writers there is a tendency to class as tuberculous all spontaneous and post-febrile broncho-pneumonias in children, but this seems scarcely consistent with our present clinical and anatomical knowledge.

[1] Loc. cit. [2] Loc. cit.
[3] Jahrbuch für Kinderheilkunde, Bd. XXIV. [4] Les Bactéries, Paris, 1888.

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RARE FORMS OF CARDIAC THROMBI.

By WILLIAM OSLER, M. D.

We meet in the heart chambers thrombi of the following forms:

First: globular thrombi, with sub-trabecular ramifications, which are common in the auricular apendices and in the apices of the ventricles in cases of extreme dilatation.

Second: mural thrombi, usually laminated, which occur in the dilated auricles, particularly their apendices, in the ventricles in cases of fibrous myocarditis, and in aneurism of the heart.

Third: pedunculated polyp-like thrombi—a very rare form—met with usually in the auricles.

Fourth: ball-thrombi, free in the auricles, which constitute the rarest form of cardiac thrombus.

The first form, the *végétations globuleuses* of Lænnec, occurs not infrequently, and is well known. The polyp-like thrombi are very rare. Hertz[1] has collected nine cases in the literature. I have never met with an instance.

The second variety, mural, laminated thrombi, are not very uncommon, and the case here reported is of interest chiefly on account of the enormous size of the thrombus.

Ball-thrombi, free in the chambers, are excessively rare, only five cases having been recorded.

CASE I.—*Large ball-thrombus, free in left auricle; mitral stenosis.*

M. S., aged 35, admitted to Montreal General Hospital, February 8th.

For twenty years she had been subject to attacks of shortness of breath, which within the past three years had become much worse. Two years ago she had an attack of acute rheumatism, and during the past eight years she had occasionally spat blood.

In 1878 she had an attack of right hemiplegia, with aphasia. Speech returned in a few days, but the hemiplegia persisted for some

[1] Deutsches Archiv. für klin. Medicin, Bd. XXXVII.

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months. Within a year she had a second attack, since which time the paralysis has persisted. When admitted there was orthopnoea; face suffused; no dropsy; complete right hemiplegia; the heart's action was very irregular; pulse 112, rapid and feeble; the cardiac dullness was increased; the apex beat was normal in situation. There was a blowing systolic murmur in mitral region. Temperature 101°. The urine contained 50 per cent. of albumen. The patient became rapidly worse; cyanosis increased, and death took place on the 13th.

Autopsy.—Small, well nourished woman; ecchymoses on face and extremities. Heart large, distended with blood clots, those in the right chamber dark in color and pulpy. The left auricle was greatly enlarged, and contained fluid blood and clots. Among these was a ball-thrombus, ovoid in shape, the size of a small pullet-egg, measuring 3.5 x 2.5 cms. It was quite unattached, and lay free above the mitral orifice. It was firm and elastic to the touch, and on the surface greyish brown in color, and presented little linear fibrinous elevations, but no roughened spot as if it had been adherent to the wall. It gave an indistinct sense of fluctuation as if central softening had occurred.[1] There were no mural thrombi in the chamber. The endocardium was opaque, and the walls thickened. The right chambers were greatly dilated; the tricuspid orifice measured 12 cms. in circumference. The auricular face of the valve presented fresh vegetations, many of them pedunculated. The walls of the ventricle were greatly thickened. The mitral orifice was very narrow, just admitting the tip of the little finger; from the auricle it looked like a small button-hole. At the bottom there was a funnel-shaped depression. The edges of the orifice were thick, of cartilaginous consistence, and were fringed with small vegetations. The chordae tendineae were short, particularly those from the anterior muscle which was attached almost directly upon the flap. The left ventricle was small, the walls over 12 mm. in thickness. The aortic valves were opaque, and presented a row of vegetations.

The lungs were crepitant throughout. They were tough and brownish-red in color; they did not contain an excessive amount of fluid.

[1] The specimen is preserved in the Museum of the Medical Faculty, McGill University.

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The spleen weighed 150 gms., and was very firm—no infarcts.

The right kidney presented several old cicatrics. In the left internal capsule were spots of old softening.

Of this remarkable form of thrombus, Hertz[1] has reported two cases, and refers to a third. In both of his cases there was mitral valve disease, and the ball-thrombus was found in the left auricle. One measured 2½ cms. in diameter, the other 4 cms. Both were rounded, and had a firm, elastic consistence. They were made up of fibrin externally, with a yellowish, granular, central portion, evidently the result of the softening, which so commonly takes place in cardiac thrombi.

The third case, which he abstracts, is reported by Macleod, in the Edinburgh Medical Journal, 1882–83. In this case a young man, aged 27, had symptoms of cholera, and on the fifth day was seized with convulsions, cyanosis and intense dyspnoea. Death occurred in about 48 hours. There was found in the right auricle a dense grayish-yellow, freely movable clot, half the size of a walnut, which lay above the tricuspid orifice.

In the same volume of the Deutsches Archiv. für klin. Medicin, Prof. von Recklinghausen states that he had first described[2] these ball-thrombi, of which he had seen two instances, both in connection with mitral stenosis. The thrombi were round, about the size of small walnuts, and lay free in the left auricle.

These remarkable structures are, as Recklinghausen suggests, globular thrombi detached from the auricular appendix, and, being too large to pass through the narrowed mitral orifice, are kept rotating in the auricle, growing constantly by the accretion of fresh layers of fibrin. It is not likely that they produce any special symptoms.

CASE II.—*Mitral stenosis of extreme grade. Enormous dilatation of left auricle by large laminated thrombus.*

Mary J. E., white, aged 48, admitted to the Johns Hopkins Hospital, on the evening of June 20th, 1889, with dropsy and extreme dyspnoea. Married and has had five children, all of whom died when young.

[1] Loc. cit.

[2] Algemeine Pathologie des Kreislaufs, Deutsche Chirurgie, Lief, 2 and 3.

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Family history negative.

She does not think that she has had scarlet fever. Has never had acute rheumatism. She has never been a very strong woman; has had shortness of breath at times for ten or twelve years. For six weeks the shortness of breath has been worse. A week ago she began to have a cough for the first time. She has been in bed for eight days, and her feet have become swollen. She has had a good deal of vomiting.

21st. Present condition. Small, spare woman; color ashy-grey; finger tips and lips blue. There is marked orthopnoea. The feet and legs are swollen. When admitted the pulse was scarcely to be felt at the wrist, and she was given two hypodermics of ether and digitalis. This morning the pulse can just be felt, but cannot be counted. Respiration 44.

Heart. Very slight visible impulse. To the hand, marked shock, especially in epigastrium. There is an indistinct thrill. Dullness extends from the upper border of third rib, and to the right is two finger's breadth beyond the margin of sternum.

On auscultation, below the nipple there is a continuous rapidly succeeding series of sounds, the first and second not distinguishable from each other, and the long pause is absent. *There is no murmur.* At lower sternum the first sound is distinguishable. It has a ringing, echoing character. At the base the second sound can be distinguished from the first, and is loudest at left margin of sternum. Most careful auscultation fails to detect a murmur at any of the cardiac areas.

She was ordered hypodermics of ether and tincture of digitalis every three hours. In the evening she seemed somewhat better, the distress of breathing was not so extreme. There was dullness at the left base, as high as the angle of scapula, with feeble breathing.

22nd. The pulse can scarcely be felt. In the mitral area the first sound can be distinguished from the second, and the diastolic pause is more marked. *There is no murmur.* The second left at base is ringing. On palpation the shock in lower sternum is very marked. The discrepancy between the loud, clear ringing sounds, with the moderately forcible impulse of the heart, and the extremely shabby, scarcely detectable pulse is very marked. The urine is scanty and difficult to obtain; it contains a trace of albumen.

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The cyanosis became more intense. She got much worse during the night and died on the morning of the 23rd.

A diagnosis of mitral stenosis was made. The absence of murmur was thought to be due to the condition of enormous dilatation of the left auricle.

Autopsy, by Dr. Welch. Multiform ecchymoses on the skin; œdema of the legs; face cyanosed.

In peritoneum, about 150 cc., of clear yellowish serum.

Thorax. The right pleura was everywhere adherent; the left pleura contained 1,500 cc. of serum.

Lungs. The right main pulmonary artery entering the lower lobe was occluded by a firm, greyish-red thrombus which extended only a short distance into the branches of the artery. There was well marked brown induration of the organ, with desquamative heart-pneumonia. The substance was dry. In the left lung the left upper branch of the pulmonary artery was completely occluded by a greyish-red, laminated thrombus. The pulmonary artery and its branches in both lungs were extensively atheromatous. The substance of the left lung was much compressed; it was also in a state of brown induration. There were no infarcts.

Heart weighed 16 oz. (453.6 gms.) (due largely to enormous thrombus in the left auricle). The left ventricle was not hypertrophied or dilated. It appeared to be normal in size, and measured 9 cms. in length. The walls were 1 cm. in thickness. The aortic valves were slightly thickened along the lines of closure; otherwise they were normal. The mitral orifice was extremely stenosed. The segments were completely and firmly united to each other everywhere, except at the aortic extremity of the orifice, where there was an opening measuring about 5 mm. in diameter, which scarcely admitted a small lead pencil. The united segments were thickened and calcified, but the surface was not rough, saving to a little extent on the auricular face. There were no vegetations. The chordae tendineae were thickened, and the tips of the papillary muscles fibroid. The left auricle was greatly enlarged, measuring 10 cms. transversely and 7 cms. vertically. The muscle wall was greatly hypertrophied, measuring 6 mm. in thickness. The endocardium was thickened and opaque. The chamber was nearly filled with an ante-mortem thrombus, laminated, partly grey and partly red. Over a greater part of its extent it was firmly

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adherent to the wall of the auricle; in other places it was loosely adherent. The thrombus partly occluded the mouths of the pulmonary veins, but there were channels through which the blood could flow. The thrombus had undergone softening in various parts. The pericardial surface of the left auricle was thickened and opaque. The right ventricle was markedly hypertrophied and dilated. It was 9 cms. in length; the walls averaged 7 mm. in thickness. The muscular trabeculae were thickened, and the tricuspid orifice admitted readily four fingers. The segments of the valve were normal, saving a little diffuse fibroid thickening. The right auricle was also much hypertrophied and dilated; its walls measured in places 4–5 m.m. in thickness. The hypertrophy was especially well marked in the trabeculae. The cavity of this chamber was much dilated, measuring at the longest about 8 cms. The coronary sinus was greatly dilated. The pulmonary valves were normal. The pulmonary artery presented several opaque yellow atheromatous patches. At its bifurcation there was a parietal thrombus, which became an occluding thrombus in the vessels going to the left lower and to the right upper lobes, as already described.

The spleen weighed 3¼ oz., (92.14 gms.) dark-red in color.

The kidneys presented patches of atrophy on the surface. The striae of the cortex were distinct. The consistence of the organs was increased. The renal arteries were atheromatous. The liver weighed 33 oz., (935.5 gms.) and was in a condition of red atrophy.

Mural thrombi are quite common, particularly in the auricular appendices, but they are usually small. Massive coagula, with extension into the vessels, such as existed in this case, are extremely rare, and occur chiefly with mitral stenosis. Cases are on record in which the thrombus has passed through the narrowed mitral orifice.

Clinically the case is interesting as illustrating the disappearance of the murmurs in the last stage of mitral stenosis, not an uncommon event when the left auricle becomes over distended. There were no symptoms which could be directly referred to the blocking of the auricle with thrombi, none which we do not meet with in extreme grades of dilatation of this chamber.

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NOTE ON ENDOCARDITIS IN PHTHISIS.

By WILLIAM OSLER, M. D.

Within the past few years several writers have called attention to the frequent occurrence of vegetations on the heart valves in phthisis. The appearance of Dr. Percy Kidd's paper in the St. Bartholomew's Hospital Reports,[1] and the dissection of a recent case suggested a review of my post-mortems, as I had the impression that the condition was by no means so common as he had found it. The following case presents points of special clinical interest.

Stella D., aged 19, admitted to the Johns Hopkins Hospital, November 1st, with cough, loss of flesh and high fever. Temperature on admission 105°.

Her mother died of consumption. She had always been well until her present illness which began about six months ago with cough and fever. Through the summer she lost flesh and had night sweats. She gave up work in May. On admission the temperature was high, and she was emaciated and anæmic. There were cavernous signs at both apices, most extensive at the left; fine crepitant râles and moist sounds at bases.

Heart. Cardiac pulsation visible in third, fourth, fifth and sixth interspaces. In the third, fourth and fifth, the impulse was wavy. At apex the heart sounds were clear. At the pulmonary cartilage there was a short systolic murmur, quite localized, not transmitted to the left and not heard below fourth rib. At the aortic cartilage the sounds were clear. The patient had persistent high temperature, reaching twice to 105°. She rapidly failed and died on the 22d.

The autopsy showed cavities at both apices. The left upper lobe was small and did not cover the heart to the usual extent. There were numerous groups of tubercles throughout all the lobes and many areas of gelatinous infiltration.

[1] Vol. XXIII.

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The heart was large. The cavities contained fresh coagula and a little fluid blood. The valves on the right side were normal. The mitral orifice was of medium size. The edges of the auricular surface were uniformly studded with large, recent vegetations, grayish-white in color, soft and readily removable. The majority of them were pedunculated. The aortic valves were free.

There were no infarctions.

Fresh frozen sections showed:—(1) marked proliferation at the attached edge of the vegetation; (2) A finely granular substance, composing the great part of the granulations,—the granules were uniform in size; (3) Scattered about among them were numerous large compound granular corpuscles, some of which were rounded, others irregular spindles.

Bacilli were not found in the stained sections.

Clinically this case illustrates the well-known fact that a murmur of maximum intensity in the 2d and 3d left interspaces may indicate mitral insufficiency, but I do not remember ever to have heard one so localized in these regions, and inaudible at the apex. I regarded it as an instance of the murmur so common at the left sternal margin in cases of phthisis and thought to originate in the pulmonary artery, but the condition of the valves would indicate that it was produced at the mitral orifice.

In 216 autopsies on phthisis there were 12 instances with fresh endocardial vegetations—mitral valves, 8; aortic valves, 3; aortic and mitral, 1. With one exception the disease was of the verrucose or warty variety, and did not produce any destruction of the segments. The case of the ulcerative form was in a woman aged 28, who was admitted January 8, 1883, to the Montreal General Hospital, under Dr. Molson, with well-marked chronic phthisis and delusional insanity. There was involvement of the greater part of the right lung, with cough, night-sweats, and rapid emaciation. There was nothing in her history to call attention to cardiac trouble, and the condition found at the autopsy was unexpected. There were extensive ulcerative changes on the mitral valves.

In Kidd's series there were twenty-seven cases of endocarditis in five hundred phthisical subjects; in thirteen of the simple warty variety, in nine associated with sclerotic changes, and in four there were chronic endocardial changes alone. In one instance the recent

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affection was ulcerative. About the same percentage was affected in his series as in mine.

The tubercle bacillus has been found in these vegetations by several observers (Cornil. Heller, Centralbl. f. Bacteriologie, 1887, 1), but it may be doubted, in the absence of the characteristic changes associated with the organism whether this is anything more than an accidental contamination. In the cases which I have examined they were not present, and Dr. Councilman tells me that he has not been more fortunate. Dr. Kidd, also, has not found them in his cases.

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Volume II No. 2

THE

JOHNS HOPKINS HOSPITAL

REPORTS

CONTENTS.

I. Tubercular Peritonitis. By WILLIAM OSLER, M. D.
II. A Case of Raynaud's Disease. By H. M. THOMAS, M. D.
III. Acute Nephritis in Typhoid Fever. By WILLIAM OSLER, M. D.

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TUBERCULAR PERITONITIS.

GENERAL CONSIDERATIONS—TUBERCULAR ABDOMINAL TUMORS—CURABILITY.

By WILLIAM OSLER, M. D.

The progress of abdominal surgery during the past few years has contributed to our knowledge of tubercular peritonitis in two directions—first, in teaching us with what frequency the condition may simulate or be associated with abdominal tumor; and, second, in demonstrating the curability of a certain proportion of the cases. To a consideration of these two aspects of the subject I propose to devote the following paper, introducing their discussion by a brief summary of certain of the anatomical and clinical features of the disease.

I.—General Considerations.

Anatomically the classifications which have been made of tubercular peritonitis are not altogether satisfactory.

It is customary, and correct, to exclude the cases of scattered miliary tubercles in the diffuse infective disease and also those cases in which the peritoneal surface of tubercular ulcers is alone involved. Practically, the great differences which we see, post-mortem, in this condition result from the situation, the rate of growth of, and the degree of inflammation accompanying the tubercles, and whether there is much or little exudation—serous, purulent or hemorrhagic. The anatomi-

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cal basis in all cases is essentially the same, and the variations which we meet, though distinct and marked, are scarcely sufficient to warrant the elaborate subdivisions of this disease made by certain writers. Thus, Spillman,[1] in his excellent recent article on the subject, makes the following five divisions: *tuberculose miliáire aiguë; tuberculose ulcereuse; tuberculose fibreuse; pelvi-péritonite tuberculose; tuberculose péritonéo-pleurale.* I see no reason for the fourth and fifth groups, if we bear in mind the frequency with which the peritoneal mischief is excited by tubal disease and the liability of the pleural membranes to be involved in the process. A large proportion of the cases in the first three divisions would at some period of their evolution come in the fourth or fifth group.

In reviewing a number of post-mortems in this disease we find that they fall naturally into the first-named categories:

(1) *Acute miliary tuberculosis*, characterized by a sudden onset, a rapid development, and a serous or sero-sanguineous exudation.

(2) *Chronic caseous and ulcerating tuberculosis*, characterized by larger tuberculous growths, which tend to caseate and ulcerate, leading often to perforations between the intestinal coils, and a purulent or sero-purulent exudate, often sacculated.

(3) *Chronic fibro-tuberculosis*, in which the process may from the outset be sub-acute, or may represent the final result of the miliary form. There is little or no exudation and the tubercles are hard and pigmented.

There exists the closest analogy between tubercle as we see it on the peritoneum and as it occurs in the lung—the fresh miliary eruption, the caseous, ulcerating masses and the chronic, fibroid, pigmented nodules may be studied with equal facility in either structure.

A few practical points in the morbid anatomy may be mentioned. In many cases the process is entirely local. Thus in five of seventeen cases of which I have post-mortem notes the condition was confined to the peritoneum. Case VIII (given in another section) is an excellent illustration of this, and it will be noted also that the mesenteric glands were not affected. This local character of the disease, upon which scarcely sufficient stress has been laid, is an extremely important feature, particularly in discussing the propriety of operation, as

[1] Dictionaire Encyclopédique, Art. Péritonites.

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the outlook is much more favorable in the absence of intestinal, pulmonary or tubal complications.

The Fallopian tubes are often affected, but the proportion given by various writers differs very much. Of my series in one only of four post-mortems in women were the tubes diseased. In seven of sixteen cases from Immermann's clinic[1] these parts were involved. It is safe to say, I think that in from 30 to 40 per cent. of the cases in woman the tubes are found affected. The process is commonly confined to the distal ends and may be primary—which is usual—or is secondary to the peritoneal involvement. Gynecologists now diagnose and remove dilated tubes with such facility that we have numerous opportunities of studying primary tuberculosis of these organs. I have frequently been impressed with the wisdom of this procedure as a protective measure, on seeing large caseous tubes with miliary nodules on the peritoneal surface, since the danger of general extension in such cases is very great. Hegar's monograph[2] is a storehouse of interesting information on this subject.

A third point, worthy of attention on account of its importance as an aid in diagnosis, is the frequent involvement of the pleura. Several of the French writers on the subject have dealt very fully with this, notably Fernet[3] and Boulland,[4] and Spillman, as remarked above, makes a special sub-division to include these cases. In Boulland's list of eighty-two cases there were thirty-eight with tuberculosis of the pleura, with or without effusion.

In only three of the seventeen post-mortems, of which I have notes, was there tuberculous pleurisy,—a comparatively small proportion. In the twenty autopsies in Häne's list there were nine with pleural involvement. In twenty-five of Bristowe's[5] forty-eight cases, the pleura was affected. It is often only a dry pleurisy, occurring most frequently without pulmonary affection, and due to a direct extension through the diaphragm. The pericardium is also liable in these cases to be the seat of an adhesive tubercular inflammation.

Age.—Tubercular peritonitis occurs at all periods of life. It is common in children, in whom it is often associated with intestinal

[1] Ueber Peritonealtuberculose, Häne. Rorschach, 1889.
[2] Genitaltuberculose des Weibes. Stuttgart, 1886. [3] Quoted by Boulland.
[4] Paris Thesis, 1885. [5] Reynold's System of Medicine.

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and mesenteric disease. Full statistics dealing with its prevalence in infancy and childhood, are not available; I am sure the figures which follow, do not represent the true proportion of cases at this period of life. It is most common between the ages of twenty and forty. In old age it is rare, but it may occur even in advanced life, as in Case XXI of my series, in which extensive disease existed in a man of eighty-two. In my own cases the distribution has been as follows; Under ten, 2 cases; from ten to twenty, 1; from twenty to thirty, 4; from thirty to forty, 5; from forty to fifty, 7; from fifty to sixty, 1; and above eighty, 1. Adding to these, 69 cases of Boulland,[1] 48 cases of Häne,[2] 39 cases of Maurange,[3] (in which the age was given), and 45 cases of Fenwick,[4] makes 222 in all; and joining these figures to those of Bristowe, Hilton Fagge and Lebert,[5] 135 cases, we have a total of 357. These analyzed according to ages give, under ten, 27; between ten and twenty, 75; from twenty to thirty, 87; between thirty and forty, 71; from forty to fifty, 61; from fifty to sixty, 19; from sixty to seventy, 4; above seventy, 2.

Sex.—The disease is certainly more prevalent among females. The statistics of general hospitals and of average medical practice may perhaps show a preponderance of cases among males. This was the case in Bristowe's figures at St. Thomas' Hospital, in Fagge's at Guy's Hospital, and Fenwick's from the London Hospital, while in my series of twenty-one cases, fifteen were males. But when we go over the recent literature of the laparotomies which have been performed in this disease, we find the number of females to be largely in excess. Thus, if we take the figures of Boulland, Häne, Maurange and my own, there are 60 cases in males and 131 in females.

Race.—It is stated that the disease is more common in the negro than in the white race. Several of the leading physicians of this city have expressed themselves strongly on this subject, particularly Dr. I. E. Atkinson and Dr. W. T. Howard. Three of the four cases which have occurred at the Hospital have been in colored people, but

[1] Loc. cit.

[2] Loc. cit.

[3] Paris Thesis, 1889. De l'Intervention Chirurgicale dans la Péritonite Tuberculeuse.

[4] Lectures on some Obscure Diseases of the Abdomen. London, 1889.

[5] Quoted by Spillman, loc. cit.

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there are, so far as I know, no figures which could enable us to arrive at a definite opinion as to the relatively greater frequency of the disease among them.

Clinically it is extremely difficult to make a satisfactory classification of the cases of tubercular peritonitis, and I shall here only refer to certain special features in the mode of onset and to peculiar symptoms not, as a rule, very fully discussed.

The process may be completely *latent* and the eruption take place so slowly and so painlessly that the patient may not have presented a single symptom of abdominal disease. The condition has thus been met with in the operation for hernia, and more frequently still in association with ovarian tumor. In three of my cases it was found accidentally, and, so far as could be ascertained, there had not been special symptoms pointing to abdominal disease. Thus in Case X,[1] a man aged 40, well nourished and believed to be in good health, was admitted to the Montreal General Hospital with strangulated omental hernia. He died eighteen hours after the operation, and extensive tubercular peritonitis of the fibrous variety was found. The left pleura was also involved. In Case XI,[1] a girl, aged 18, was admitted to hospital with severe typhoid fever, of which she died. The abdominal symptoms were those ordinarily met with and there was no history of previous trouble. The post-mortem showed, in addition to characteristic typhoid lesions, an extensive tubercular peritonitis, which had taken its start from the Fallopian tubes. The lungs were not affected. Case XII,[1] a healthy looking, well-nourished child of five, died of malignant diphtheria after an illness of a few days. An acute miliary tuberculosis existed over the entire peritoneum, which contained a slight amount of serous and much fibrinous exudation. There were tubercles in the spleen but none in the lungs. A case at present in the Hospital, in Dr. Kelly's ward, illustrates this latency in the disease. The patient had a large ovarian tumor which was removed October 18th. The peritoneum was found universally covered with recent tubercles of various sizes which also existed over the surface of the tumor.

The literature contains very many cases of this kind, so that it is a fair conclusion to regard the disease in many instances as latent in its

[1] Of the series of 21 cases.

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course, and it is possible for the process to go on to healing without having induced serious symptoms.

The onset of symptoms may be *sudden* so that the diagnosis of enteritis or hernia may be made. A remarkable instance in which it was mistaken for the last-named disease is reported by Thoman.[1] A well-nourished woman, aged 30, was suddenly seized with pain in the abdomen, vomiting and fever. The physician who saw her believed the symptoms due to a hernia, which he thought he found and reduced. The condition continued and in the evening Thoman was called in. No hernia was found externally but as the abdomen was distended and painful it was decided to operate. The inguinal ring was found closed. In the further course of the disease, the peritonitis became more marked, the ascites increased and death occurred on the fourteenth day. The post-mortem showed extensive tuberculosis, both layers of the peritoneum being covered with a recent eruption. There were no tubercles in the lungs or pleuræ. This case is not unique, as Spillman[2] quotes another instance in which the symptoms were so urgent and deceptive that internal strangulation was suspected.

This suddenness of onset is very deceptive and usually leads to the diagnosis of a simple acute peritonitis. The following case which I saw on several occasions with Ross of Montreal illustrates this point as well as the importance of the pleural symptoms so liable to supervene in the course of the disease:

CASE I.—*Acute peritonitis; tympanites; abdominal tenderness, with loss of flesh and irregular fever. Pleurisy with effusion. Tubercles on peritoneum, pleura and parietal pericardium.*

G. C., aged 17, colored, was admitted to the Montreal General Hospital, January 23rd, 1884, with an attack of acute peritonitis which had begun suddenly three days before. For a year he had had irregularity of the bowels, with occasional pains, and had lost flesh. On admission, temperature was 104°, pulse 92. The abdomen was tympanitic and there was pain on pressure. Under appropriate treatment the pain became less and he improved. The temperature fell and became subfebrile, until February 1st, after which it was very irregular, rising to 102°, 103° or 104° at night, becoming normal or even subnormal

[1] All. Wiener Med. Zeitung, 1887, page 306. [2] Loc. cit.

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in the morning. Towards the end of February there was a return of the pain and tenderness in the abdomen. Early in March, signs of pleurisy on the left side and a friction murmur developed near the left nipple, thought to be pericardial. By the 17th of April, there was well-marked effusion into the left pleura. The heart impulse was felt to the right of sternum. The abdomen became more distended, a little hard; no ascites. On the 30th, 1½ litres of serum were removed from the left chest. By May 7th, the fluid had re-accumulated and he was again aspirated. Death took place on the 19th.

Autopsy.—(Case 795, Post-mortem Records of Montreal General Hospital.) Much emaciation. Abdominal organs and coils of intestines completely glued together. Peritoneum studded with innumerable whitish masses, the size of small marbles. On section they were firm and caseous. Pericardium was adherent to sternum and there were tuberculous nodules infiltrating the parietal layer. The visceral layer was smooth; no tubercles. In left pleural cavity 2 litres of fluid. The pleura was covered with tubercles; the lung was airless and lay close against the spine. There were no tubercles in the substance of either lung. The abdominal organs were normal. There were vegetations on the aortic valves. The brain was normal. Dr. Ross, in commenting on this case,[1] notes particularly the abrupt onset of the acute peritonitis, which under appropriate treatment rapidly disappeared.

The disease may set in with pronounced *gastric symptoms* and simulate ulcer or cancer, as in the following case already published in my Pathological Reports, Montreal General Hospital, 1878.

CASE II.—*Acute tubercular inflammation of the peritoneum. Persistent gastric symptoms. Small caseous mass in left lung. Right-sided pleurisy.*

J. McT., aged 35.—Had been a soldier for twelve years, latterly a sailor; admitted to Montreal General Hospital in September, 1876, complaining of weakness, loss of appetite, and frequent attacks of vomiting. No albumen in urine. Blood normal. Systolic murmur at apex. No enlargement of abdominal organs. Tenderness on deep pressure along right costal border and ensiform cartilage. The vomiting became more marked, and he had occasional attacks of diarrhœa.

[1] Canada Medical and Surgical Journal, Vol. XIII.

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The symptoms pointed, though vaguely, to disease of the stomach, either round ulcer or cancer. The vomiting was with difficulty controlled, and patient became very weak and anaemic, the skin slightly icteric. He gradually got so feeble that he was unable to move from bed, and the vomiting was so persistent as to necessitate feeding per rectum. Through January and February the vomiting diminished, but the patient wasted slowly, and the case was regarded as malignant disease, involving perhaps the peritoneum. In the beginning of May the peritonitis became acute and general, and he died on the 25th, profoundly exhausted. For some weeks before death hemorrhages occurred in various parts of the skin.

Autopsy.—The peritoneum, contained 3 litres of a turbid, slightly bloody fluid, in which were flocculi of lymph. Here and there the coils of intestines were matted together by easily separable adhesions. The transverse colon and stomach were in this way glued together; the former covered also the anterior border of the liver. The entire peritoneum, except the portion over the stomach, was of a dark red color, infiltrated, sodden, and readily stripped off from the subjacent tissues. Localized patches of lymph occurred here and there upon it. The whole membrane presented a great number of small white areas, flat, not projecting above the surface, and ranging in size from a hemp seed to a split pea. As a rule they were isolated, but occasionally groups were seen. They existed in about equal numbers over the intestines, mesentery, and parietal peritoneum. Beneath the latter were from eight to ten larger white patches, which, on section, had a caseous appearance, were firm to the touch, not encapsulated, and extended to the depth of about four millimetres. On examination of these small and large white masses, they were found to be almost entirely subperitoneal and composed of aggregations of lymphoid corpuscles, a little smaller than the colorless blood corpuscles, and with one, rarely two, nuclei. In sections through those on the intestinal wall, the corpuscles were seen to infiltrate to some extent the muscular coats. The mesenteric glands were but little enlarged.

The heart showed numerous ecchymoses on pericardium; the walls were flabby, the muscle pale; very little blood in the chambers.

There were 2 litres of turbid fluid in right pleural sac. Visceral and parietal layers congested, and covered with flakes of lymph. A few cc. of fluid in left sac.

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The right lung was crepitant, except at extreme base, the lower lobe collapsed. The organ contained a good deal of serous fluid. The left upper lobe was crepitant, the lower collapsed and œdematous. At the anterior border of upper lobe was a firm block of condensed tissue, somewhat triangular in shape, which on section was made up of a small cavity, looking not unlike a dilated tube. There were one or two caseous nodules in the lung. There were no miliary tubercles in either lung.

The spleen weighed 150 gms. unaltered.

The liver weighed 2000 gms., was anæmic and yellowish in color.

The kidneys were normal in size, but very firm in texture. In the cortex of the right were several small purulent foci, about which the substance was much congested.

The stomach did not present any trace of cicatrices or of tumor. It contained about a litre of fluid.

The small intestines contained yellowish, liquid fæces; the walls were thick, owing to an infiltrated, swollen condition of all the coats. The mucous membrane was dark in color. Peyer's glands were not enlarged.

A more common mistake is confounding tubercular peritonitis with *typhoid fever*, which it may simulate very closely. R. L. MacDonnell,[1] of Montreal, has recently called attention to this, and has reported several cases. In the following instance, also in Case IV, this mistake was, I believe, made.

CASE III.—*Attack of acute abdominal disease; gradual recovery; latent pleural effusion. Pronounced tuberculous history.*

Miss G., aged about 30, fairly well nourished, seen in October, 1888, on account of shortness of breath and cough. The family history was bad; several members had died of tuberculosis. She had herself always enjoyed fair health. In August she began to have abdominal pains and an irregular fever, in which the temperature rose as high as 102.5°. She was thought to have typhoid fever, though there was no diarrhœa and spots were not found. The most marked symptoms were the distension of the abdomen and the tenderness, chiefly on the right side. The temperature chart, which I

[1] Canadian Practitioner, 1888.

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saw, showed an irregular fever range, not at all characteristic, the temperature sometimes dropping to normal. After persisting for about six weeks, these symptoms subsided, the fever left, and she got up and began to gain strength; but shortness of breath became a marked feature and she remained pale and developed a slight cough, and it was for these symptoms that I was consulted. The abdomen was a little full, tympanitic, nowhere tender, no signs of any effusion. On inspection of the chest, it was seen that the right side scarcely moved; the apex beat was far over in the left axillary line. There were dullness and other signs of extensive exudation in the right pleura. Temperature was normal. She was aspirated with great relief and two weeks later fluid was again withdrawn. She improved rapidly and by the middle of December, the breath sounds were well heard over the greater part of the right lung, but percussion resonance was defective over the lower half, and at the base quite flat.

Considering the nature of the abdominal attack, the patient's family history and the gradual onset of the pleural effusion, there can be but little doubt that this was a case of tubercular peritonitis, mistaken for typhoid fever.

Ascites is a frequent symptom but it does not as a rule become very marked; thus Biat,[1] in an analysis of eighty-one observations, found only thirteen instances with extensive ascites. In the acute miliary tuberculosis with rapid exudation the effusion may be bloody, but judging from the published records and from my personal experience this is not so common as in cancer, though the opposite statement is usually made. It has frequently been mistaken for the effusion in connection with cirrhosis, of which, indeed, it may sometimes be a complication. It is somewhat remarkable with what frequency acute tuberculosis of the serous membranes occurs in this disease. Moroux[2] and Wagner[3] have called attention to the involvement of the peritoneum, which in my experience is not so often affected as the pleura. I have notes of six cases in which acute tubercular pleurisy occurred as a final complication in cirrhosis.

Cases with extreme *tympanites* are also common. This condition, the result of impairment of the tone of the muscular coats, is a very constant feature in all forms of the disease. There are instances in

[1] Paris Thesis, 1884.
[2] Paris Thesis, 1883.
[3] Deutsches Archiv f. Klin. Medicin, Bd. xxxiv.

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which it seems to be particularly marked, as in Case VI, to be referred to later.

Of special symptoms, I wish to speak of two only; one of which has not received the attention it deserves.

Sub-normal temperatures.—Many writers refer to the fact that the temperature in tubercular peritonitis may be normal, but it is not generally known that the temperature may be subnormal for weeks or months at a time. My attention was called to this fact about four years ago by my colleague Dr. Musser, at the Philadelphia Hospital, who has made a number of observations on this point. In the cases of fibrous tubercle, without much inflammatory process or effusion, there is as a rule very slight fever and subnormal temperatures are common. Thus, in Case VII, to be fully given under another section, the temperature during the patient's entire stay in the Hospital was subnormal for a greater part of the day. In the early morning the thermometer rarely indicated more than 96° or 96.5°; a gradual rise occurred through the day, and the normal point was reached late in the afternoon. The same was noticed in a second case, upon which laparotomy was performed, Case VIII. During her convalescence, for days at a time, the temperature did not once reach 98°; thus during November 28th, 29th, 30th and December 1st, the temperature was taken every two hours, day and night. On the 29th and 30th it ranged between 97° and 98°, but twice registered at 96°. Throughout December 1st, 2d and 3d it only once reached 98°; the range was between 96° and 97.5°. In the diagnosis of doubtful cases this symptom may prove of great value.

In a case of Sir Edward Sieveking's, at St. Mary's Hospital, London, reported by J. F. Payne,[1] the patient's temperature from March 21st to April 16th, ranged from 95.6° to 96.4°. Such references as this occur in the literature, but they are by no means common and the fact is not widely recognized.

Pigmentation.—An increase in the skin pigment, particularly on the face, is an occasional symptom in tuberculosis of the peritoneum. It was specially described by Guéneau de Mussy in 1879,[2] but I remember in the session of 1872–73, that Sir Wm. Jenner, at University College Hospital, pointed out this condition as simulating Addison's

[1] Transactions of the Pathological Society of London, Vol. xxi.

[2] Étude sur la Pigmentation de la Face dans la Tuberculose abdominaire. Paris, 1879.

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disease in a case of extensive abdominal tuberculosis. It was present in a marked degree in Case VII, and I have seen one other instance. In Case XII of Boulland's paper, the symptoms of Addison's disease were pronounced and, post-mortem, tubercles were found in the suprarenal capsules as well as on the peritoneum. I think the condition may be present when the tubercles are confined to the peritoneum, and an increase in the pigmentation does not necessarily mean that the adrenals are affected.

II.—Tumor Formations in Tubercular Peritonitis.

To the occurrence of tumor-like formations in tubercular peritonitis we are indebted for much of the increase in our knowledge on this subject, as the errors in diagnosis have shown the frequency with which these tumors occur and also how amenable the condition is to surgical treatment. The question has not been fully considered by any recent writer, yet its importance may be gathered from the fact that in 96 cases in which laparotomy was performed, in 37 the diagnosis was tumor, ovarian or otherwise.

One of the best and most suggestive, and perhaps the first, of the papers to deal with this question was by Dr. W. T. Howard, of Baltimore.[1] He reviewed the literature of ovarian disease with special reference to this point, and showed how little attention had really been paid to it; yet, even in 1885, before laparotomy had become so common for peritonitis, he was able to refer to several instances in which the mistake had been made of confounding encysted effusion with ovarian tumor. As he remarked, the standard works on gynecology did not allude to the subject, and with the exception of a brief note in Kaulich's[2] monograph, there was no reference in general medical literature. Busey[3] had previously reported a case in which an encysted peritonitis simulated ovarian cyst, and Gardner[4] an instance in which the diagnosis of a suppurating cyst was made.

More recently Van der Warker[5] reported an interesting case and discussed the propriety of laparotomy in tubercular peritonitis.

[1] Transactions of the American Gynecological Society, 1885.
[2] Prager Vierteljahrsschrift, 1871.
[3] Gaillard's Med. Journal, May, 1880. Quoted by Howard.
[4] Canada Medical and Surgical Journal, 1885.
[5] American Journal of Gynecology, 1887.

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Among the numerous monographs and papers on the operative treatment of peritonitis in recent French and German literature, I have not met with one which discusses as it deserves the question of these tumor-like formations.

We may recognize anatomically, and possibly clinically, four groups of cases in which with tubercular peritonitis tumors occur and may be felt on examination: First, omental tumor; second, sacculated exudation; third, retracted and thickened intestinal coils; fourth, mesenteric glands.

(*a.*) *Omental Tumors.*

On the thin and delicate layers of the epiploon tubercles will be found if present at all on the peritoneum, but they do not often form large masses which can be felt through the abdominal wall. The omental tumor in connection with this form of peritonitis results from a slow tubercular process which gradually puckers and rolls the membrane, until it forms an elongated firm mass attached to the transverse colon lying athwart the upper part of the abdomen. This condition, perfectly well recognized by clinicians, is in many cases peculiar and distinctive. I call to mind at least four instances, in two of which the diagnosis was confirmed post-mortem. Of these, two did not occur in my own practice. In the third, a man at the University Hospital, Philadelphia, with a tuberculous history and symptoms which pointed to gastric trouble, the abdomen was moderately distended, painless, and there lay across the upper zone a ridge-like tumor, readily separated from the liver and spleen. It was not possible to exclude cancer but the diagnosis leaned rather to tuberculosis, and this was confirmed some months after on the death of the patient, which took place outside the Hospital. The fourth case is of interest, as it occurred in a man over eighty, who presented simply a condition of general enfeeblement with moderate wasting and slight enlargement of the abdomen. Here, too, there was an elongated mass in the upper part of the umbilical region, which proved on post-mortem to be a solid omental tumor caused by chronic tubercular inflammation. There were no tubercules in the lungs or pleuræ in this case, which illustrates also a condition which is more common than is supposed, viz: tubercular infection in the aged.

These cases often occur without much exudation and result from

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a slow, latent process which may run its course without exciting serious symptoms. To diagnose this condition from cancer is often difficult. A pronounced tubercular history, subnormal temperatures —which are not I think so common in cancer, and which are specially likely to occur in these more chronic cases of tuberculosis—and the existence of disease in the pleuræ or lungs are suggestive indications. The impossibility of avoiding error is illustrated by a case of Gairdner's[1] in which an omental tumor was thought to be mesenteric. In connection with the subject this writer says that "it would be easy to show that in most of the text books the diagnostic characters and significance of thickening of the great omentum have been strangely overlooked; although the mere anatomical fact has long been known."[2] Cases I and IV in the appendix to his lectures are of special value as indicating the gradual resolution in children of these tubercular omental tumors.

Fagge[3] calls attention to the existence of a resonant percussion note above the mass, which sometimes feels as if attached to, and indeed has been mistaken for, the edge of the liver roughened and nodular. This point is of some importance in the diagnosis of the omental tumor. It must be remembered, too, that when the mass lies close to, or even upon, a distended colon firm percussion may elicit flat tympany. Crozer Griffith[4] has recently reported an interesting case in a man, aged 64, in whom a tuberculous omental tumor complicated rather than simplified the diagnosis.

R. L. MacDonnell has given me the notes of the following case in which the omental mass formed a prominent tumor in the right iliac and lumbar region—an unusual situation.

Emma S., aged 30, admitted to the Montreal General Hospital, April 5th, 1887. A thin delicate woman with a history of scarlet fever at fourteen, rheumatism at seventeen, followed by some pulmonary trouble. She had always been subject to constipation, neuralgia and general indisposition. Some years ago she had a foot

[1] Loc. cit.

[2] The justness of this criticism is appreciated after a perusal of Péan's large work (Tumeurs de l'abdomen, 1880, Tome I) in which he devotes, of twenty-eight pages, just a half a page to tubercle of the omentum and does not even allude to this most common form of tumor.

[3] Practice of Medicine. [4] University Medical Magazine, October, 1888.

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amputated on account of disease of the ankle joint. About three weeks previous to admission, her present illness began with an attack of severe pain in the abdomen. With this she had vomiting, particularly after taking food. The bowels were obstinately constipated. On admission there was severe abdominal pain; dorsal decubitus, with knees drawn up; pulse small and frequent; temperature normal; tongue heavily coated; abdomen distended, no fluctuation, no tenderness, except in the left lumbar region over the descending colon. Heart and lungs negative.

May 3rd. The condition remained practically the same. Much pain and tenderness over the abdomen. A hard tumor could be plainly felt in the right iliac and lumbar regions, lying quite to the right of the middle line. She was removed to a private hospital and Dr. Gardner performed laparotomy. A large tubercular mass was found in the omentum, occupying the position above noted, in some places it was adherent to the intestines. A fæcal fistula resulted and she died of exhaustion.

Klebs[1] describes an extensive fibro-caseous thickening of the peritoneum which in one case formed a dense, opaque yellow mass a hand's breath in width attached to the parietal layer and stretched across the abdomen just below the navel. Such a mass might be readily confounded with an omental tumor. Fenwick says[2] that the thickened capsule of the spleen may produce a tumor-like body in the left hypochondriac region. More common I should think would be the tumors associated with thickening of the capsule of the liver to which he also refers. Here an exudation sacculated between the capsule of an enlarged liver—and, as Strümpell notes, this condition is not uncommon in tubercular peritonitis—and the anterior abdominal wall may produce a localized tumor of great distinctness.

There might possibly be in tubercular disease a cystic accumulation within the layers of the great omentum. Péan[3] cites such a case in which there was an enormous tumor in front of the intestine, containing a brownish semi-purulent fluid and gas, the walls of which were evidently formed by the layers of the omentum. Obliteration of the foramen of Winslow by tumor or by chronic peritonitis has been followed by encysted hydrops between the epiplooic layers.

[1] Handbuch der Pathologischen Anatomie, Berlin, 1869.
[2] Loc. cit.
[3] Loc. cit., p. 338.

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(*b.*) *Sacculated Exudations.*

These are the most common, as they are undoubtedly the most puzzling of the abdominal tumors produced by tuberculous disease; so puzzling, indeed, that, as a long list of cases shows in which the operation for ovariatomy has been performed, the very elect among gynæcologists may be deceived.

In these cases a sero-fibrinous or purulent exudation is confined and limited by adhesions formed between the intestinal coils, the parietal peritoneum, the mesentery and the abdominal or the pelvic organs. What is felt as tumor may be entirely fluid or it may have an irregular nodular character from the presence between the coils of large caseous masses.

These sacculated tumors, due to tuberclosis, may, as in other forms of peritonitis, be met with in the upper, middle or lower abdominal regions. In the upper zone, which includes the stomach, liver and spleen, encysted collections of fluid are extremely common. Thus, we have the localized peritonitis associated with gall bladder disease, and with various affections of the stomach and of the liver and spleen. The effusion in these cases may be limited entirely to the upper region of the peritoneum. In the tubercular disease by far the most common sacculated exudation occurs here with peri-hepatitis, and as in the case of Emma G. (p. 99) over the surface of an enlarged liver, may lead to the suspicion of a gall-bladder tumor projecting below the edge of the ribs. I think, however, from an analysis of the cases, that these encysted peritoneal tumors are less common in the upper abdominal region.

In the middle zone, which includes the peritoneal cavity from the level of the transverse meso-colon to the false pelvis, and which embraces the omentum and intestine, these encysted tumors are much more common and as the record of operations shows are very frequently mistaken for ovarian tumor. In reviewing a list of such cases, it seems that they fall into two divisions, those in which the entire anterior portion of the peritoneal cavity was occupied by a large collection of fluid and those in which a more limited sacculated exudation was found on one or the other side of the abdomen or in the middle line. The following remarkable case reported by Gardner,[1] of Montreal, illustrates the former :—

[1] Canada Medical and Surgical Journal, Vol. XIII.

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A. B., aged 23, unmarried, was sent to him by Dr. Ross for examination, as there had been a suspicion of pregnancy. She could give no definite account of the date at which the present abdominal enlargement began, but it had been noticed for three or four months and had rapidly increased. There was pain in the abdomen; the general health and strength had declined and she had become emaciated. The menses had been absent for three months.

Examination.—The abdomen was much enlarged; well marked fluctuation in the anterior and antero-lateral aspects, with dullness on purcussion in these areas. In the flanks and epigastrium, the bowel note was present; no firm or solid part to be felt anywhere. The anterior aspect of the abdomen was quite uniform. The uterus, measuring two inches, was pressed upwards and forwards. The patient was admitted to the General Hospital, when it was found that she had fever of a septic type, the temperature running very high. In the centre of the anterior part of the abdominal wall about the navel, there was œdema and a red blush. The diagnosis of suppurating ovarian cyst was made. At the operation, on reaching the peritoneum, no separation of parietal from visceral layer could be made. The knife entered a collection of fluid, passing through what seemed to be a thickened, closely adherent cyst wall. The cyst was drained and irrigated. The general condition improved for ten days. The temperature then rose and she developed a cough with purulent expectoration. She sank rapidly and died six weeks after the operation.

Autopsy.—(No. 825, Post-mortem Records, Montreal General Hospital). Moderately emaciated girl. A two-inch wound between the navel and the pubes contained a drainage tube which passed through Douglas' fossa into the vagina. On opening the cavity of the peritoneum, a large mass the size of a man's head, occupied the false pelvis. This and the parietes were covered by a grey, rough membrane half an inch in thickness. The transverse colon, although firmly adherent upon the surface was also bent upon the liver. Drawing this mass and the liver towards the right side, a collection of pus was found below and by the side of the spleen, and another small collection lay under the left lobe of the liver. On careful examination it was found that the anterior peritoneal cavity was converted into a suppurating cyst, extending from the liver to the pubes. The pelvis was nearly filled by the globular mass referred to above.

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This consisted of all the intestines except the transverse colon, closely matted together by recent soft adhesions, which were studded with miliary tubercles. Everywhere the walls of the cyst appeared older than the internal adhesions and had all the appearance of unhealthy granulating membrane. The walls and viscera of the true pelvis were covered with the same membrane.

The lungs contained many grey tubercles but no cavities. Both lungs were universally adherent.

The intestines were normal.

In this remarkable case the sac occupied a large part of the peritoneal cavity and pushed the intestine into the pelvis. It is interesting to note the œdema and redness about the navel, at which point in these cases of tubercular peritonitis spontaneous perforations sometimes occur.

These large purulent exudations simulating ovarian tumor are not necessarily tubercular but, as in the remarkable case described by Dr. Ewing Mears,[1] may be puerperal.

In a larger number of these cases the tumor is more localized and either lateral or central in position, and it may be quite impossible to make a diagnosis from developing ovarian tumor. The following is a case of the kind to which additional interest is added by the gradual and complete disappearance of the tumor.

CASE IV.—*Illness simulating typhoid fever; development of an abdominal tumor which gradually disappeared. Rapid pulmonary tuberculosis.*

Early in November, 1884, I was consulted by a young lady from Montreal, from whose statements and from the account of her physician, R. L. MacDonnell, the following history was obtained: No tuberculosis in the family; she had, though somewhat delicate, enjoyed average health. Early in June she was confined to bed with a low fever, thought to be typhoid. The temperature ranged from 100° to 102°. The abdomen gradually became distended. By the end of July it was evident that there was fluid in the peritoneum. The dullness, however, was not movable but persisted in the left iliac and right lumbar regions when the patient was turned on her

[1] Transactions of the College of Physicians, Philadelphia, 1875.

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right side. The urine became very copious in amount, of low specific gravity—1005. The question was discussed whether the patient had not an ovarian tumor with mild typhoid fever. Throughout August she improved. The abdomen diminished in size and became irregular in outline. The left side gradually became more prominent. On the 26th of August, Mr. Lawson Tait examined the patient and said that the tumor might be one of three things: tubercular peritonitis, parovarium cyst or a congenital subperitoneal cyst, working its way up in front of the abdomen. He predicted gradual absorption and recommended incision and drainage when the fluid became thickened. Throughout September she improved very much and the tumor reduced in size. On September 20th, Dr. MacDonnell noted that "beyond a doubt an encysted tumor, as large as an adult head, lay on the left side of the abdomen." I saw the patient on the 10th of November. The abdominal symptoms had almost disappeared and there was left nothing more than an obscure sense of fullness and thickening in the left side. I could scarcely believe from the examination that there had been the large tumor described and sketched by Dr. MacDonnell. The lung symptoms were marked and the patient was rapidly failing. She returned to her home and died in December.

The majority of the cases in which encysted effusions have been mistaken for ovarian tumor, have been of this kind. The exudation is sacculated either between the intestinal coils, in which case it may be deep-seated and give only a sense of obscure fluctuation or, as is more usual, the parietal peritoneum forms the anterior wall of the sac and the collection simulates an ovarian cyst.

Lastly, there are the sacculated exudations within the pelvis proper in which case the disease almost always starts from the Fallopian tubes. The tubercular process may be exclusively upon the parietal peritoneum and the coils of intestines glued to the lateral walls may shut off completely the pelvic from the general cavity.

(c.) *Retracted and thickened intestinal coils.*

The matting together and thickening of several coils of the intestines may form a mass of great distinctness and even lead to the diagnosis of a solid tumor. This is most frequently met with in the

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cæcal region. They are not necessarily fixed tumors but may be freely movable as in Case IV of Spaeth's paper.[1] The following case is a good illustration:

CASE V.—*Tumor in right iliac region, believed to be malignant. Gradual loss of flesh and strength. Pain and diarrhœa. Tumor formed of intestinal coils in cæcal region.*

Man aged 48. For twelve months he had had pain in the right lumbar region and in the right groin. Micturition was frequent and he had occasional diarrhœa. He had not passed blood in the stools, and there had been neither obstruction nor vomiting. There was a well-marked tumor in the right iliac region. Towards the end, a cough with muco-purulent expectoration developed and there was dullness at the base of the right lung. During the last three days of life the abdomen was tender and tympanitic. The tumor was believed to be malignant in character.

Autopsy.—Slight emaciation. Abdomen distended, 500 cc. of turbid fluid, with many flakes of lymph in the peritoneum. The coils of the intestines were matted together, but could be separated. A mass of adherent bowel filled up the right iliac fossa; several of the coils communicated with the colon, by perforations in the terminal part of the ileum. These coils and the ascending colon formed a firm solid mass which occupied the right iliac fossa and which had been mistaken during life for a tumor.

When slit open the lower two inches of the ileum were found to be extensively diseased. The walls were thickened and the mucosa ulcerated. The cæcum was much contracted, only admitting the thumb. The wall was nearly half an inch in thickness. In the upper part of the ileum there was a typical tuberculous ulcer. There was general tubercular peritonitis, and the serous covering of the liver was greatly thickened.

The lungs presented small cavities at the apices surrounded by fibrous tissue and groups of tubercles. The lower lobes were normal. The kidneys presented a few small tubercles.

Here no doubt the starting point of the trouble was in the cæcum, in which the disease was much more extensive and older looking

[1] Deutsche Med. Wochenschrift, No. 20, 1889.

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than in any other part. The solidity and firmness of these tumor masses formed by the intestinal coils are very remarkable and as in this case are very apt to lead to error in diagnosis.

There is another remarkable form of intestinal tumor, the result of chronic peritonitis, not necessarily tubercular. The small intestine is shortened by puckering and thickening of the mesentery. The walls are enormously thickened and the entire coil may form a firm knot, lying close against the spine. When matted together by adhesions this coil of intestines may give on examination the idea of a solid mass. The following is a remarkable instance of the kind:

Sarah A., aged 82, admitted to the Philadelphia Hospital, December 22, 1887, with ascites, stated to be of several months duration. At first the effusion was moderate but it increased so that tapping was necessary. About five litres of a sero-fibrinous fluid were removed. The liver could not be felt, but presented about three inches of vertical dullness in the nipple line.

The spleen was not palpable. After withdrawal of the fluid, a rounded, firm mass about the size of a cocoanut, could be felt, and seen, in the central part of the abdomen. It was somewhat movable and a little irregular on the surface. The fluid reaccumulated and she was again tapped and an equal amount withdrawn. The tumor was centrally placed, and so readily separated from any of the abdominal viscera that I thought it very probably of retro-peritoneal origin.

The autopsy, January 26th, 1888, showed the peritoneum covered with flakes of moderately firm lymph. The tumor was seen to be made up of the small intestine greatly shortened and thickened, the coils closely united with each other forming a mass the size of a large cocoanut, closely adherent to the spine. It seemed scarcely credible that the small intestine, even puckered and thickened as it was, should form so firm and so small a mass. The mesentery was very greatly thickened. There was much pigmentation of the peritoneal coat, the muscular wall was greatly thickened and the mucous membrane of the ileum was thrown into thick folds, resembling the valvulæ conniventes. The transverse colon and sigmoid flexure were much contracted. There was thickening about the appendix and in the mucous membrane of the cæcum there were two small ulcers. The liver presented senile atrophy but no cirrhosis. There was extensive perihepatitis.

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The other organs presented no special changes.

Tubercles were not found, and the case appears to have been one of chronic peritonitis, starting possibly from the cæcal region.

A very similar condition to this has been found in the chronic tubercular disease. Some years ago I performed a post-mortem for Howard, of Montreal, on a woman aged about 30, who had signs of chronic disease of the peritoneum with ascites. On opening the abdomen, the entire cavity was converted into a large fæcal abscess. The anterior wall of the cæcum was completely destroyed by the tubercular ulceration and the fluid fæces had passed directly into the peritoneal cavity. The small intestine formed a puckered and retracted coil which lay close against the spine, forming a firm bunch which, as in the other case, presented a strange appearance in contrast to the greatly distended peritoneal cavity.

Prochownick[1] reports a remarkable case in a girl of 16, who presented in the right side of the abdomen a hard somewhat nodular tumor, which extended from Poupart's ligament to a point above the navel. At the operation the mass was found to be composed of the entire intestinal tract, from duodenum to the beginning of the rectum, united in a single coil, closely matted together and covered with lymphoid granulations.

The coils may not form, as in these cases, a uniform tumor, but there may be a separation into three or four irregular masses, divided by fissures and covered with thick lymph.

It is possible for the coil to form a resonant tumor; thus Goodell writes that "in one of his cases of tubercular peritonitis the intestines were gathered up towards the sternum in a bag of false membrane, making a well-defined resonant tumor, which was very puzzling until the abdominal cavity was opened."[2]

(*d.*) *Mesenteric Glands.*

Less common, perhaps, in tubercular peritonitis than any one of the previous conditions is the presence of tumors caused by enlarged glands. So far as I can ascertain, in none of the cases of laparotomy did they lead to an error in diagnosis. Cases are, however, on record

[1] Deutsche Med. Wochenschrift, No. 24, 1889.

[2] Private letter, November 29th, 1889.

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in which extensive tuberculosis of these glands formed palpable tumors, associated with ascites. One of the most accurately described is by Gairdner:[1] A man, aged 21, had nodular tumors "of varying distinctness, sometimes nearly superficial, sometimes over-lapped by intestines, not capable of being identified with any of the greater viscera, to a great extent mobile and chiefly felt in the left umbilical region, very dense, hard, irregular and somewhat nodulate, altogether having much of the position and some of the characters of mesenteric glandular tumors." The post-mortem showed these glands greatly enlarged, hard, inelastic; on section solid, no suppuration but presenting a yellow infiltrated matter. There was no disease of the lungs or of other organs, nor was the peritoneum involved. One cannot doubt the tuberculous nature of this case. I have not seen in the adult a similar one in which the tuberculosis, confined to the mesenteric glands, produced large tumors, but I have seen a precisely similar condition limited to the retro-peritoneal glands. (Philadelphia Hospital, Post-mortem Records, 1888, p. 220.) These are cases of the abdominal scrofula of the old writers. Bamberger[2] gives an instance, the only one in his experience, in which a woman, aged 60, who had vomiting, diarrhœa and signs of marasmus, presented nodular masses in the abdomen, above the navel, which were mistaken for gastric cancer. Post-mortem showed extensive tuberculous infiltration of the mesenteric glands without tubercles in other organs.

Besnier[3] states that Colin has described three cases in soldiers, in whom were found enormous tubercular tumors of the mesenteric glands without ulcers in the intestines.

Andral[4] records the case of a man, aged 29, with ascites, and enlarged cervical glands, in whom, with extensive tuberculous peritonitis, an enormous tumor was found, due to infiltration of the glands. Occasionally in phthisis there is great enlargement of these bodies without any indications during life. Such a case was in the Philadelphia Hospital last year (Post-mortem Records, 1888–89, p. 60), with enormous enlargement of the mesenteric glands, forming large irregular tumors. Tympanites may mask this condition, as in a case given by Henoch[5] in which a mesenteric tumor, the size of the child's

[1] Loc. cit.
[2] Virchow's Handbuch, Bd. VI, p. 708.
[3] Dictionaire Encyclopédique. Article Mesentery.
[4] Clinique Medicale, T. II, p. 648, 1839.
[5] Loc. cit.

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head, was entirely concealed by the distension of the intestines. Sometimes, after the removal of the ascitic fluid, or when it is in slight amount, there can be felt irregular nodular bodies or cord-like thickenings of great distinctness, and it is not always feasible to determine whether these are glandular or large caseous masses between the coils of intestines.

A question of special interest relates to the association of mesenteric gland disease with tubercular peritonitis. Gairdner, in the lectures already referred to, has urged that in a large proportion of the cases of so-called tabes mesenterica, in which there is enlargement and hardness of the abdomen—the condition which the French speak of as *carreau*—there is involvement of the peritoneum. Jacobi has recently expressed the same opinion.[1]

The *diagnosis* of these peritoneal tubercular tumors offers difficulties which vary greatly in the different varieties. The omental tumor is probably a less frequent source of error than any other, but as an identically similar condition may exist in cancer, it is not always possible, unless there is marked tubercular disease elsewhere, to determine the precise nature; and, as we have seen, even an acknowledged expert like Gairdner may be led astray.

The lumpy, nodular character of the mesenteric tumors gives to them also a certain degree of distinctness. The mistake is sometimes made, nor do I think it can always be avoided, of confounding the large caseous nodules situated between the intestinal coils with the mesenteric glands. The possibility of their recognition depends very much on the degree of distention of the bowels, as extreme tympanites may completely cloak a very large tumor of this character.

The tumors formed by contracted and thickened intestinal coils usually lead to error in diagnosis, nor do I see, save in most exceptional circumstances, that this could be avoided.

The recognition of the saccular exudation, more particularly its differentiation from cystic ovarian disease, offers really serious difficulties, the extent of which may best be appreciated by the fact that of 96 cases of laparotomy in tubercular peritonitis, in not less than 30 ovarian disease was supposed to be present. Such being the case, it may be worth while to discuss briefly certain diagnositic details.

[1] New York Medical Journal; II, 1889.

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There is no single criterion which enables us to say in a given case that the condition is one of encysted peritonitis, nor indeed is there any special group of symptoms which can be regarded as distinctive. It were folly to lay down, in parallel columns, differential rules in an affection in which again and again the ablest diagnosticians in our profession have erred.

It will suffice merely to touch upon the points most suggestive, in individual cases, of tubercular trouble:

First. The history of the patient and of the disease. Tubercular antecedents are common. Evidence may exist of old tubercular lesions. Gradual failure in health and strength may perhaps be taken into consideration, but it must not be forgotten that in many of the cases the patients have beon robust and well-nourished. The mode of onset is in the majority of instances gradual, but this is such a variable factor that it is not of very much value; perhaps the most which can be said on this point is that there can usually be elicited a history of obscure abdominal pains, irregular febrile attacks and altogether a greater degree of gastro-intestinal disturbance than generally accompanies the slow evolution of ovarian cysts. If the case has been under observation for some time, the fever record should be of great assistance, as high or very low temperatures more commonly occur in this condition, though it is true that in inflamed and suppurating ovarian cyst there may be fever of a hectic type.

Second. The local physical signs. If possible, these are more deceptive than the history and symptoms. The question is not so much between the characters of a sacculated exudation and ascites, but it is the extremely nice one of discriminating between two varieties of sacculated effusion, ovarian and peritoneal. In typical cases, the physical signs have conformed in every particular to those of cystic ovarian disease. There are a few indications which may at times be useful; thus when the sacculated tumor is limited and small the outlines may not be so definite and clear as in ovarian disease. This is a point referred to by several writers. The position and form may be variable owing to alterations in the calibre of the surrounding intestinal coils of which in part the walls are composed. At the periphery of the tumor irregular, nodular bodies —cheesy masses—may sometimes be felt, which in several instances have led to the diagnosis of malignant disease. Depression of the

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vaginal wall is not a safe indication one way or the other, as I find the condition mentioned as present in ovarian tumor as well as in encysted peritonitis.

Third. In every case the condition of the tubes and of the lungs and pleura should be most thoroughly examined. The association of a tubal tumor with an ill-defined, anomalous mass in the abdominal cavity should arouse suspicion at once. So also the evidence of involvement of the pleura or of the apex of one lung. It is rather surprising, in looking over the reports of cases, how little attention seems to have been paid to these most important and common concomitants of tubercular peritonitis.

III.—The Curability of Tubercular Peritonitis.

Until within the past few years, the general opinion in the profession has been that this disease is incurable; and in looking over the text-books of medicine, with but few exceptions—Fagge a notable one—the prognosis is given, as in the words of Flint, "always fatal." Henoch,[1] in his admirable account of this affection in children, says that when recovery has followed in certain cases in his practice, he has thought the diagnosis incorrect, and that the peritonitis had really been of the simple chronic form. Yet there exist not a few reports among the older writers, indicating that a form of chronic peritonitis, not to be distinguished from the tubercular, did occasionally get well. More recently McCall Anderson,[2] of Glasgow, in a clinical lecture published in 1877, reported three cases illustrating recovery in tubercular peritonitis. The history and the symptoms left no doubt as to the correctness of the diagnosis, but the cases were regarded as altogether unique. Gee,[3] in 1881, stated "that recovery from tubercular peritonitis is common." Gairdner[4] also has insisted upon the occasional cure in this affection, while admitting that there was a hiatus in our knowledge of the changes undergone in the progress towards healing. Ashby,[5] in his article on peritonitis in children, says "a large number of cases completely recover." Fenwick, in his recent lectures,[6] speaks less hopefully of permanent cure. The evidence

[1] Vorlesungen ueber Kinderkrankheiten, 4te Auflage, 1889. [2] Lancet, 1877.
[3] Lancet, Jan. 1st, 1881. [4] Loc. cit.
[5] Cyclopedia of Diseases of Children. Edited by Keating. Vol. III, 1890.
[6] Loc. cit.

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has been rapidly accumulating to show that in a considerable number of cases, recovery in this disease is possible, either spontaneously or after operative interference.

(*a.*) *Spontaneous Cure.*

There is no inherent improbability why tubercles on the peritoneum should not undergo involution as they do elsewhere. Anatomically the peritoneal growth bears in its evolution a close analogy to the pulmonary, and this is still further borne out by the retrograde changes through which it passes. Just as the aggregations of miliary nodules in the lung may undergo the changes which we speak of as healing, becoming hard and fibroid, so in the peritoneum the tubercle tends in many cases to become sclerotic, and passes into a condition in which it is practically harmless. This beneficial result is more likely to be seen in cases belonging to the third group, in which, from the outset, the process is sub-acute and not associated with much exudation; but there are cases on record in which recovery has followed even after extensive effusion.

The anatomical changes are, in brief, these: fibroid and pigmentary induration of the tubercles, absorption of the exudate, transformation of the fibrinous material into connective tissue, with the union to a greater or lesser extent of the intestinal coils and of the peritoneal surfaces with each other. The following case illustrates this condition:

CASE VI.—*Chronic tympanites, with constipation; gradual development of pulmonary symptoms, death; adhesive peritonitis with fibroid tubercles. Pulmonary tuberculosis.*

W. C., age 38, colored, admitted to the University Hospital, Philadelphia on the 18th of January, 1888. His father died of phthisis. He had been a healthy laboring man of temperate habits. He had not had any serious illness. About four weeks before admission he first noticed distension of the abdomen and he found that it was difficult to button his clothes. For some time he had been very constipated.

Following note was made on admission: "The patient is fairly well nourished, says he has not lost in weight, complains of swollen abdomen and constipation; temperature 101.3°; pulse 82. On examination uniform enlargement of the abdomen, measuring 72 cm.; respira-

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tion is costo-abdominal. On palpation, soft, no special resistance; percussion gives resonance anteriorly and in the flanks; in illiac regions resonance not so full; there is no dullness, no percussion wave. Neither liver nor spleen palpable. Area of liver dullness diminished; two inches of vertical splenic dullness." The case was regarded for a time as one of ordinary tympanitic distention, associated with sluggish and constipated bowels. The persistent elevation of temperature ranging from 99° to 102° and slight tenderness in the flanks, with recurring night sweats, aroused a suspicion of tubercular trouble, but the examination of the lungs was negative. Throughout the month of February, there was a daily elevation of from 1 to 3 degrees; he lost flesh and began to cough; there was, however, no expectoration, but examination determined a few scattered râles, most marked at the left apex, behind. During March he continued to lose in weight, the sweats were less troublesome, the temperature rarely rose above 101°; the condition of the abdomen remained the same; there was slight tenderness in the flanks; no dullness; measurement about 75 cm.; no signs of effusion could at any time be discovered; the local disease had at the left apex behind become more marked, the breathing was slightly tubular and there were numerous mucous râles. The constipation remained a marked feature, the bowels were never moved without a purge or an enema. He gradually failed without any further development of the pulmonary symptoms, and death occurred on the 19th of March. The post-mortem showed disease of the left lung, partly old, with slight fibroid change and many recent tubercles and cheesey masses. In the abdomen, the peritoneum was obliterated by universal adhesions between the layers. The coils of small intestines were united together by old fibrinous bands; here and there in the adhesion were pigmentations and small, hard, dark tubercles. Numerous adhesions existed over the liver, uniting it strongly to the diaphragm, and in these, too, there were many old fibroid tubercles.

Here the peritoneal disease was practically cured, but the ill effects remained in the weakening of the intestines. The pulmonary not the abdominal affection caused death.

Similar cases might be drawn from the records of any pathologist of large experience. In Cases XIII and XIV of my series,

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an identical condition existed. In both, death took place from pulmonary disease, and the peritoneum presented universal adhesions in which were hard, deeply-pigmented fibroid tubercles.

In this connection the cases which Gairdner gives in the appendix to his lectures are of the greatest interest, as three of them illustrate this gradual improvement in undoubted tubercular disease. In Case I, a child, aged 8, presented signs of peritoneal disease, with moderate effusion, and later, thickening of the great omentum. The improvement within three months was remarkable, though slight induration of the omentum remained. The improvement continued and two years subsequently the patient was well. In Case III, the improvement was also most striking under simple treatment; the effusion disappeared, but evidence of omental thickening persisted. Case IV is still more remarkable. A child, aged 9, presented well-marked thickening of the omentum, and other symptoms pointing to peritoneal involvement. During the two years she was under observation, the general health improved and a gradual resolution of the omental tumor took place.

No writer has dealt with this aspect of the question, so fully and clearly as Boulland.[1] He has ransacked the literature of the subject, and in his collection of eighty-one cases in which tubercle occurred in the pleuro-peritoneal membranes, there are at least twenty cases of peritoneal tuberculosis in which recovery took place. He places the number of recoveries much higher than this, but I have excluded many doubtful cases on his list. In many instances, of course, this may have been only a temporary improvement, but in three instances quoted from Bucquoy, the good health persisted ten, twelve and seventeen years after recovery. The subsequent history of operative cases removes all grounds for skepticism—reasonable perhaps a few years ago—as to the genuineness of these cases. One of the most interesting of the cases quoted by Boulland is from Louis. A man aged 24, with great enlargement of the abdomen, signs of pleural affection and extreme marasmus, was attacked with Asiatic cholera. He nearly died from the excessive purging, but the abdominal effusion disappeared and he ultimately made a good recovery. In reading the details of the long list of cases given by Boulland, one receives the impression that the cure of tubercular peritonitis cannot be a very

[1] Loc. cit.

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uncommon event. Case III, to which I have already referred, as simulating typhoid, is an illustration, I have no doubt, of a clinical group by no means rare. The following is an interesting example of marked improvement in, if not actual healing of peritoneal tuberculosis:

CASE VII.—*History of an obscure abdominal affection with fever and loss of flesh. Gradual improvement. Ill-defined abdominal tumor. Local disease of the lungs. Pigmentation of the skin. Marked improvement.*

A. B., aged 31, merchant, admitted to the Johns Hopkins Hospital May 18th, 1889, complaining of swelling and distress in the abdomen, with weakness and loss of flesh.

Family history is good. Father and mother living and healthy; two brothers living; two sisters died when children.

He had dysentery 15 years ago and with that exception has always enjoyed good health until October, 1888, when, after exposure to cold, he had an attack of obscure trouble in the abdomen. There were swelling, tenderness and a sense of distension and weight, particularly in the region of the liver. There was no diarrhœa, rather constipation. He lost flesh and became extremely weak. At Christmas he was up and about, and in January attended to his business. The strained, distressed feeling in abdomen persisted. Throughout February and March he remained pretty well, though far from his usual condition of health. The swelling of the abdomen subsided greatly. Early in April the distention increased again so that he could not button his trousers, but he had neither pain, diarrhœa, nor fever. He again lost flesh rapidly.

Condition on admission: Large boned man, 5 ft. 11½ in height; marked emaciation; orbital fat much wasted; eyes sunken, with deep, dark rings about them; cheeks very hollow. The forehead, cheeks and chin were distinctly pigmented; this darkening in color he had noticed gradually coming on since October. The skin of abdomen and backs of hands were also pigmented. The chest was large; ribs prominent. Expansion was deficient at left base. Percussion was clear with the exception of the left base, where the resonance was slightly defective and here fremitus was diminished. There was also slightly defective resonance at right apex and the right clavicle was more prominent than the left. On auscultation there was feeble breathing

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with fine râles in the lower axillary and infra-scapular regions. The contrast between the two sides was most marked. At the right apex there were moist sounds heard just beneath the clavicle, and behind in the upper part of inter-scapular area.

Abdomen was moderately distended, uniform. No fluid discoverable. Flanks were tympanitic. The resonance was defective just below and to the left of the navel, in an area equal in size to the palm of the hand. On palpation, no tenderness, but in the region below and to the left of the navel there was an ill-defined, tumor-like mass, resistant, not painful but slightly tender on deep pressure. It was readily separable from liver and spleen. Below, the margin was well defined.

Liver dullness not increased. Edge of spleen not palpable. Inguinal glands not enlarged. No tenderness in renal regions. The temperature was subnormal. Urine negative, not increased in amount. He had slight morning expectoration, muco-purulent in character, which did not contain tubercle bacilli, but in which once elastic tissue was found.

Patient improved rapidly in Hospital. Abdomen reduced in size. He gained in weight. June 6th he went to Atlantic City, where he improved in a remarkable manner. He returned on the 19th, having gained over 25 pounds since his first entrance to Hospital. He had no cough. The color had improved and he had lost the characteristic abdominal facies. The examination of the lungs showed that the note at the left base was clearer; there was a dry friction rub in the lower left axillary region. Râles still persisted at the apex. The abdomen was a little full, though not so much so as at first. There was a distinct prominence to the left of the navel, and here the same obscure tumor-like mass could be felt. In the right inguinal region about two inches above Poupart ligament, there was also a distinct, ridge-like projection which was not noticed at previous examinations.

The temperature range of this patient during his stay in the Hospital was very carefully studied. For days at a time two hourly observations were made. Unfortunately the charts were mislaid, but the general results may be stated as follows: The range was between 96° and 99°. The temperature fell throughout the morning hours, and by 6 or 8 A. M. reached the minimum, then gradually rose through the fore-

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noon and only reached a normal point in the late afternoon hours. The persistent low temperature was one of the most striking features of the case.

The patient returned to his home in a Western State, and has remained well ever since. He was seen by Dr. Toulmin in October, when the condition of good health persisted.

The attack in 1888, with swelling and tenderness of the abdomen and loss of flesh, was, without question, I should say, tuberculosis of the peritoneum. The existence of the tumor-like mass, the subnormal temperature curve, the signs of involvement of the pleura and of the lung, the diffuse pigmentation—all point to the existence of this affection. The striking improvement which occurred throughout May and June has persisted, and I see no reason why a permanent cure should not be established.

The cases which are most likely to terminate favorably are those in which the infection is limited to the peritoneum, the inflammation of moderate grade and the effusion slight in amount and sero-fibrinous. The instances I have given in illustration of the latency of the disease would seem to indicate that an adhesive inflammation, as it is termed, may accompany the process from the outset, and that a gradual sclerosis may overtake the tubercles and render them harmless. Caseation and ulceration, with a sero-purulent exudation, preclude the possibility of spontaneous cure. Extension to the pleura and lungs and the co-existence of intestinal or tubal disease are conditions equally unfavorable to permanent recovery.

(*b.*) *Cure by Operation.*

The beneficial effects which, in a number of cases, followed the opening of the peritoneum when a sacculated exudation was mistaken for ovarian tumor, encouraged surgeons to perform laparotomy in ordinary cases of tubercular peritonitis accompanied with much effusion. Sir Spencer Wells, in 1862, performed laparotomy on a patient believed to be the subject of ovarian tumor, but in whom the condition was found to be tubercular peritonitis. The effusion was withdrawn and the patient recovered. She married and at last report, twenty-five years after the operation, maintained her good health. The operation, thus unintentionally carried out in this and many

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subsequent cases, was advocated strongly by Hegar[1] and by Koenig[2] in Germany, by Lawson Tait in England, and has since been practiced by many surgeons in Europe; and in this country by Homans of Boston, Mundé of New York, Goodell of Philadelphia, Kelly of Baltimore, Gardner of Montreal, and others.

In two cases recently under treatment in the Hospital, laparotomy has been performed, in one for tubercular peritonitis, though a doubt existed whether or not a tumor was present; in the other an ovarian tumor was found accidentally to be complicated with a latent peritonitis. The first case presents features of very special interest, as rapid amelioration followed the removal of the fluid, while the death from acute disease, after convalescence was established, enabled us to study the changes in the peritoneum which are associated with the healing of tubercular processes.

CASE VIII.—*History of obscure abdominal trouble for several months; acute exacerbation with high fever. Doubtful abdominal tumor. Laparotomy, drainage; rapid improvement. Discharged, feeling well. Return with acute pneumonia, death. Chronic tubercular peritonitis in process of healing. Syphilis of rectum. Amyloid liver.*

Emma G., age 28, admitted August 28th, complaining of pains in abdomen.

Father and mother probably living, one brother and one sister living; two sisters and two brothers dead, none of them of lung disease. Had whooping-cough and measles as a child; ten years ago had pleuro-pneumonia, was ill five months and has not been perfectly well since. For five years she has had a cough, on and off, and on several occasions has spat blood; has had no special illness since the attack ten years ago, but several times in the spring or autumn she has been in bed with weakness and shortness of breath. Her present trouble began about six months ago with swelling of abdomen, which has been variable in extent and has on several occasions almost disappeared. She has generally been constipated, has been short of breath and has had palpitation of the heart. She had been working up to a few days before applying to Hospital. On admission the following note was made: "Temperature 101°. Patient is a well-

[1] Loc. cit. [2] Centralblatt für Chirurgie, 1884.

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grown, not emaciated woman. The tongue is coated white. The abdomen is much distended, measures 86 centimetres; it is symmetrical, extremely tender to touch in the upper zone, particularly below the right costal margin. The lower zone is more flaccid and less tender. On percussion, when lying on back the flanks are dull, umbilical region resonant; on changing posture, flanks become resonant and the fluid gravitates towards the centre of abdomen. The liver dullness reaches from fifth rib to nearly two inches below costal margin. Both percussion and palpation in this region very difficult on account of the exquisite tenderness in epigastrium. There is dullness from the ensiform cartilage 7 cm. downward. There are nodes on the tibiæ. Inguinal glands slightly enlarged. The heart sounds are normal. Lungs, percussion clear anteriorly; resonance is defective at the extreme base on the right side; there is tenderness here on palpation." The temperature range until September the 4th was from 100° to 103°.

Both Dr. Lafleur, who first saw the case, and Dr. Atkinson, who joined him in consultation, regarded it as one of peritonitis. There was, however, a doubt about the extremely tender area below the right costal margin, and here Dr. Halsted thought there were indications of a distinct tumor, possibly a gall bladder. On the 4th an exploratory laparotomy was made. The incision extended from the costal border 2.5 cm. to the right of median line. The peritoneum was found filled with a bloody serum. The liver seemed enlarged. The capsule was studded with tuberculous nodules. The intestines were matted together and the layers of the peritoneum presented tubercules. The cavity of the abdomen was drained and then washed out with sterilized salt solution. Histological examination by Dr. Councilman showed the tuberculous character of the peritoneal growths.

The temperature on the evening of the 4th was 104°. She rallied well from the operation. The temperature fluctuated from 99° to 102° until the 14th, when it fell to 98°, from which date until the 30th the range was from 98° to 100°. Her general condition improved very rapidly and she was up at the end of a month. She had no cough and expressed herself much better. She was about the ward, and on the 22d of October, six weeks after the operation, the following note was made: "General condition continues good; she is

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up every day ; the abdomen is still a little distended, and on palpation is tender on right side, and there is here between costal margin and illiac region a well-defined firm swelling, slightly resistant and very tender. On percussion there is tympany in umbilical and left lateral regions almost to the back ; to the right there is dullness from 5 cm. beyond the navel. On firmer percussion flat tympany can be elicited except in the extreme right flank where it is dull." She continued to improve through November, and on December 12th she was discharged. The following note was made :

" Patient went out to-day feeling quite well. All signs of tumor have disappeared ; the abdomen is soft, but in the right hypochondriac region a little more resistant than elsewhere ; here, too, it is now tympanitic."

The temperature had been normal and sub-normal for weeks.

The patient was re-admitted January 8th, with fever and urgent dyspœna.

She stated that she had been at work since her discharge. On December 30th, she had a slight chill, followed by fever and cough. On January 4th, she had another chill, with quite high fever, and on that day went to bed. On admission her temperature was 102°, respiration 60, pulse 120 ; physical signs showed an extensive area of consolidation in the right lower lobe, which extended anteriorly to the nipple line and as high as the fourth rib. The sputum was mucopurulent, a little blood-tinged and contained numerous pneumococci but no tubercle baccilli. The abdomen was not specially distended, but was quite tender in the upper zone. On the 11th and 12th she seemed better but the physical signs persisted. Temperature was not high, never reaching above 102.5°. On the 14th, she passed blood in the stool, and in the evening she had a profuse hemorrhage from the bowels and died at 10.20 p. m.

The following is a condensed report from the autopsy record by Dr. Councilman : A well-built, well-nourished woman. A smooth cicatrix extended diagonally across the abdomen, 14 cm. in length. Pigmented macular scars over the entire body, more marked on the anterior surface.

Peritoneum adherent to anterior abdominal wall over the liver. A few slight adhesions with the omentum. The adhesions over the liver

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were firm, and contained a good deal of fat. Here and there in the adhesions were firm, whitish nodules, which varied in size from a pin's head up to 3 mm. in diameter. The omentum was thickened, its upper surface smooth, its lower surface covered with numerous small, up to 2 mm. in size, firm, nodules, especially numerous along the thickened inferior border. Most of these were pigmented. No adhesions between intestinal coils. Over peritoneal surface, numerous small, firm nodules slightly pigmented. Many of these were seated flat on the peritoneum, others in small connective tissue bands, attached by one end to the serous surface. The tubercles were generally seated at the end of these. They extended, with about the same frequency, along the entire length of small intestine, but were most numerous for 75 cm. above the valve. The large intestine on its surface contained very few of these nodules. The mesentery contained numerous tubercles, partly seated on the membrane, partly along the intestinal border, in a few places matted together. The surface surrounding them was thickened and puckered as though from slight cicatricial formation. Nearly all of these contained in the tubercle dark pigment. The posterior surface contained a few, and generally smaller tubercles than on the omentum.

In the right pleural sac, 400 cc. of purulent fluid with flakes of fibrin. The pericardial membranes were adherent, slightly thickened but presented no evidence of either tubercle or caseation. The heart showed no special changes.

Lungs. The left was bound down by old, tolerably firm adhesions. The tissue was crepitant. Muco-pus could be squeezed from the small bronchi. Right lung slightly adherent at base, somewhat compressed by the pleuritic exudation. The entire pleural surface covered with fresh exudation which could be stripped off. The lower lobe, the middle lobe and part of the upper lobe were solidified. Cut surface smooth and reddish in color, and from it a reddish fluid could be squeezed. The posterior parts of the upper and middle lobes were grayer in color than elsewhere. Portions excised sank in water. The bronchial glands large, pigmented, not caseous.

Liver was large, weighed 2910 grammes; the entire surface, especially the upper, was covered with adhesions, in which and in the capsule there were numerous tubercles, either single firm nodules or flattened masses. Even when these were apparently situated in

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the liver surface they could be stripped off with the capsule, which was very much thickened. In reality this was not the capsule, but a thickened and newly formed connective tissue membrane over the entire organ.

Spleen. Capsule slightly thickened and presented numerous adhesions, in most of which were small nodules. Kidneys showed no special changes. The mesenteric glands were enlarged, firm, whitish-gray in color, a few of them slightly pigmented. No caseation. No tubercles. Small intestines showed no special change. In the rectum there was a loss of substance encircling the entire bowel with much cicatricial tissue about it and two recent-looking, deeper ulcers from which apparently the hæmorrhage had come. No tuberculosis of the tubes.

The microscopical examination showed the liver to be intensely amyloid. The tubercles in the peritoneum were composed of numbers of sub-miliary nodules, very fibrous, containing few cells in a firm tissue. In the middle of the masses, giant cells with mural nuclei and fatty granules; no caseation in the nodules. Tubercle bacilli were very abundant.

This case presented in turn many points of interest to the physician, the surgeon and the pathologist. In the first place it is a good example of primary tuberculosis of the peritoneum; not even in the pleura or pericardium, both of which showed old adhesive inflammation, were there granulations, and a most rigid search failed to find tubercles elsewhere. Surgically, the operation was a success as the symptoms were relieved, the general health improved and she left the Hospital looking and feeling well. Then the accident of an acute pneumonia gave an opportunity of studying the condition of the peritoneum four months after an acute exacerbation and showed the tubercles undergoing fibroid change but still retaining their characteristic structure and still very rich in bacilli.

The other case illustrates the latency of peritoneal tuberculosis and the extent which it may reach before inducing serious symptoms.

CASE IX.—*Gradual swelling of abdomen; tumor on left side. Ovariotomy. Extensive tubercular peritonitis; recovery.*

Bridget N., age 42, admitted to the gynæcological ward October 17th. Married 19 years, has had 8 children. Has been ill, on and

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off, ever since the birth of her last child 5 years ago. Has had metrorrhagia. She has had successive attacks of abdominal swelling, and within the past year has noticed a lump on the left side, which has gradually grown larger, and it is for this she sought relief. On inquiry she states that every winter she is apt to have a cough, and has had pleurisy on the right side. Her mother died of pulmonary hemorrhages. Other members of her family healthy.

Dr. Kelly operated October 18th; removed a tumor of the left ovary, the size of a cocoanut. On the right side the ovary was as large as a lemon, cystic and the tube greatly dilated. The ovarian tumor and the entire peritoneum, visceral and parietal, were covered with miliary tubercles. The intestines were in places matted together. There were 500 cc. of fluid in the peritoneum. The tubercular nature of the growths was demonstrated microscopically. The nodules were firm and hard, some of them pigmented. She did well after the operation and was sitting up out of bed by the 6th of November, with the wound perfectly healed. The temperature ranged for the first ten days from 98° to 100°. After the 28th the range was between 98° and 99.5°. On Dec. 18th the following note was made: "She has gained in flesh and looks well. The abdomen is a little distended but is not tender and there are no signs of effusion. There are dullness, râles and feeble breathing at the base of the right lung where she has had pleurisy. The apices of the lungs are clear."

The statistics showing the results of this operation have lately been collected by several writers, particularly Kuemmel[1] and Maurange.[2] This last writer has made an elaborate analysis of the cases recorded to date, seventy-one in all, with the following result: There died after the operation, six; by generalization of the tubercle, seven; there recovered sixteen cases, of which no further mention than this fact was made; fifteen cases were alive at the end of six months, and twenty-eight cases had survived a year.

Of the American cases, Maurange includes those of Homans (3), of Van der Warker (1), Morrill and Bradford (1), Cabot (2), Goodell (1), and Bruen (1). To these I can add the following cases. Goodell writes[3] that he has operated upon four cases, in all of which the

[1] Archiv. f. klinische Chirurgie, 1888. Bd. XXXVII.
[2] Thèse de Paris, 1889.
[3] Private letter, Nov. 28th, 1889.

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ascitic fluid was recognized, but in addition ovarian disease was suspected. In none of the cases were the tubes or ovaries diseased. So far as he could ascertain, all of the cases got well. One of them, however, after six months excellent health, returned with a pelvic tumor and ascites. She refused an operation and is now probably dead.

Mundé writes[1] that he has operated on three cases, in all of which there was ascites, and the diagnosis of obscure tumor was made. In one case the patient recovered from the operation and died two months later of pulmonary disease, which was not evident at the time of the operation. In the other two cases, the recovery was temporary and they died afterwards of exhaustion. In all three, a distinct abdominal tumor appeared to exist; in two general, in the third in the left ovarian region. There was no doubt in any of the cases as to their tuberculous nature.

Kelly has operated upon four cases.[2] The first case was in 1886. There was temporary improvement. Some months after a second operation was performed. The patient is at present alive and well. The operation in this case was performed for tubal disease and peritonitis was found. In the second case there was no definite diagnosis but a tumor mass was evident. An encysted purulent peritonitis was found which was drained. Patient recovered temporarily and died one year after of phthisis. In the third case, the diagnosis was a parovarian cyst. The first operation was in May, 1889. The abdomen was drained, improvement followed for a time, but the fluid re-accumulated and on three subsequent occasions, at intervals of about six weeks, the peritoneum was incised and drained. At the last operation the tube and ovary of the right side were removed. The patient is still under observation and has evidence of some fluid remaining in the peritoneum. The fourth case has already been referred to, in which the tubercular disease was found as an accidental complication with an ovarian tumor.

Homans'[3] fourth case, operated upon March 19th, 1889, left the Massachusetts General Hospital in June quite well. His second case, included in Maurange's statistics, operated upon April 20th, 1887—referred to at page 44 of his statistical account of three hundred and

[1] Private letter, Nov. 18th, 1889.
[2] From notes given by Dr. Robb.
[3] Private letter, November, 1889.

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forty-four laparotomies[1]—is of great interest, as Dr. Cutler examined the peritoneal growth and found it to be tubercular. In May, 1889, more than two years after the operation, she remained perfectly well.

Two cases operated upon by Gardner, of Montreal, have already been referred to in previous sections of this paper (pp. 80, 83.)

H. P. C. Wilson, of Baltimore,[2] has operated upon one case in which the disease was thought to be a cystic ovarian tumor. The entire peritoneum was studded with miliary tubercles. The patient recovered from the operation but died six months afterwards.

Dudley, of Chicago,[3] operated upon one case in 1884. There was double ovarian disease as well. She recovered but a fistula remained. Death occurred in 1888. G. E. Shoemaker[4] reports a case of recovery.

To these cases, for statistical purposes, may be added the four reported by Spaeth,[5] as they are not referred to in Maurange's paper. Of these, one died after the operation; the second, three months after of acute phthisis; the third, four months after of tuberculosis of the intestines, and the fourth, at the time of report, had intestinal disease. M. Schmidt[6] has reported two cases, one of which recovered completely and was well more than a year after the operation, the second was benefited temporarily but death occurred five months after. Imlach[7] states that he has had five cases, all of which had resulted in apparent cure.

Of these additional twenty-six cases, the results cannot be said to be on the whole so satisfactory, as fourteen cases were dead at the time of the report, one of an intercurrent pneumonia.

The majority of writers on the subject speak hopefully of the operation in suitable cases, and from what we know of the natural history of the disease and from a study of the cases in which laparotomy has been performed, whether specifically for tubercular disease, or by accident, we may regard it as not only justifiable but urgently indicated in many cases.

Secheyron[8] concludes from an analyses of forty-two cases of lapa-

[1] Boston. Sawyer & Sons, 1887.
[2] Private letter, Nov. 11th, 1889.
[3] American Journal of Obstetrics, Nov., 1889.
[4] Medical and Surgical Reporter, April 13th, 1889.
[5] Loc. cit.
[6] Centralblatt f. Gynäcologie, 1889, No. 32.
[7] British Med. Journal, Dec. 14, 1889.
[8] Nouvelles Archives d'Obstétrique et de Gynécologie, No. 11, 1887, quoted in American Journal of Obstetrics, Vol. 21, p. 447.

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rotomy, that interference is not called for in the acute or chronic disease when generalized, whether with or without sero-purulent effusion, and thinks that the operation is only called for when symptoms of strangulation or of perforation of the intestines appear. He acknowledges that encysted tubercular peritonitis calls for surgical intervention as the condition is really one of cold abscess.

Spaeth, too, does not write very encouragingly, but in a disease heretofore believed to be incurable the statistics of Maurange show such a percentage of recoveries, that we may place the operation among the triumphs of recent surgery.

Two questions remain for consideration, what cases are most suitable for operation, and how can we explain the beneficial influence?

Undoubtedly the cases of the first group, those with fresh eruption and considerable effusion, whether free or sacculated, offer the best chance of recovery, as the disease is more likely to be primary in the peritoneum, the general condition is usually better, and the subsequent chances of general infection are much slighter. When the Fallopian tubes are extensively diseased, and when the process has extended through the diaphragm to the pleura, the condition is of course less favorable. The existence of marked omental tumor, in the form of a transverse ridge, need not necessarily be an objection to operation, as we have seen that in two of Gairdner's cases, spontaneous resolution of such masses took place. In cases then with somewhat sudden onset, rapid development of ascites with fever of moderate grade, we may be most sanguine of success.

In the class of cases with extensive caseous masses in the peritoneum and a purulent exudation, the outlook is necessarily less hopeful, but even in such instances, particularly when the exudation is sacculated, laparotomy may be advised as a palliative measure.

In the chronic adhesive form, no benefit could be expected to follow the operation, which could only be intended to remove an omental mass or to open a sacculated effusion. In the majority of the cases of this group nature is effecting a cure in which she scarcely needs outside assistance; and the danger lies not so much in the peritoneal disease as in the risk of pulmonary affection.

It is difficult to explain the beneficial results of the operation. It is interesting to note that not alone in tubercular peritonitis, but in

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other forms with effusion, the simple opening and drainage of the cavity has seemed to exercise a very beneficial effect on the subsequent course of the disease. Thus, Homans reports a case[1] in which an exploratory laparotomy was performed in a woman, aged sixty, with enormous ascites: Forty pounds of fluid were removed and a soft tumor was found attached to the sacrum and right ilium. The abdomen was sponged out and sewed up, as it was found impossible to remove the tumor. The patient recovered rapidly, was greatly relieved and the fluid never re-accumulated. Death occurred a year subsequently, and at the autopsy a sarcoma was found filling the pelvis. This would indicate that the thorough drainage of an ascites, even of enormous extent, may so alter the condition of the peritoneum that the fluid is not re-formed. More remarkable still are the cases which indicate that the mere opening of the abdominal cavity modifies in some way the development of new growths. Gairdner states[2] that Sir Spencer Wells informed him of a case of apparently cancerous peritonitis, in which, after an exploratory incision, the symptoms subsided and the woman got well. Mr. Lawson Tait[3] comments at some length on this remarkable tendency of abdominal neoplasms to undergo retrograde changes after an exploratory incision.

His statements on this point are most interesting and deserve the careful consideration of physicians as well as surgeons. He says that he has seen tumors disappear after laparotomy in cases of disease of the liver, spleen and head of the pancreas. He does not specifically mention cancer of the peritoneum. His remarks deserve quoting, as they bear directly upon this subject.

"The cases are far too numerous, and the results indicate sequence far too clearly, for us to dismiss the phenomena as a mere coincidence; nor can we accept the explanation of subsequent medical treatment as having brought about this much-desired ending. I am satisfied that the mere opening of the peritoneal cavity has a direct influence in setting up the process of absorption of the tumor, and my conviction in this direction has increased my confidence in the principle of exploration. That some emphatic physiological change is at once set up by opening the peritoneal cavity is clearly

[1] Loc. cit., page 40.
[2] Loc. cit., page 46.
[3] Edinburgh Medical Journal, Nov. and Dec., 1889.

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indicated by the uniform onset of a most distressing thirst, which lasts for days, and is not seen so markedly after any other operation known to me. Let the incision in the abdominal wall be made down to the peritoneum, but let the serous cavity remain unopened and this thirst is not marked. But let the peritoneum be opened but a finger's breath and the result is marked. That a therapeutic change is effected in the peritoneum itself by the mere opening of the cavity is now universally recognized in the treatment of what we call tubercular peritonitis by abdominal section. I have now had a large experience on this point, and can say positively that we can cure permanently and speedily cases that have gone even as far as suppuration, by opening and cleansing. But in the bad cases in all probability the cleansing is never complete, no matter how much time and care are spent on it. And, in the non-purulent cases, I very often do no cleansing at all, but merely empty out the serum and put in a drainage-pipe. Yet the great majority of these cases are cured by these simple means."

Evidently, in whatever way brought about, the opening and drainage of the peritoneum favors in a remarkable way the regression of the tubercles; and it does more than this, for, as has been frequently noted and as is well indicated in the history of Case VIII, with an improvement in the local symptoms the fever reduces and the general condition of the patient rapidly improves. In some way the operation renders the condition of the peritoneum more favorable to the fibroid changes by which alone healing is induced.

There are on record several cases from which we may get an idea of the condition of the peritoneum some months after the operation. The case of Emma G., so often referred to, is probably as good an example as could be obtained of healing tuberculosis. The effusion had disappeared, in the neighborhood of the liver the adhesions had become fibroid, the tubercles were hard and pigmented, and there was nowhere any congestion about them. In the literature there are several cases of this kind. Thus, Hirschberg[1] narrates the case of a woman upon whom laparotomy was performed for peritoneal tuberculosis, and in whom the abdominal symptoms subsided completely. Eight months after, the patient died of phthisis, and there were no traces of the numerous granulations which had existed on

[1] Quoted by Kuemmel. Loc. cit.

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the membrane at the time of the operation. Another case, also quoted by Kuemmel, a girl aged 17 with ascites and tubercles in the peritoneum died six months after the operation. The autopsy showed that the effusion had not been reproduced and that healing had occurred.

A third case of Ahlfeld's[1] is still more interesting, in which he found, during the performance of Freund's operation, the peritoneum covered with granulations. At the autopsy, a year and a half afterwards, there was no trace of the tubercles.

Several views have been advanced in explanation of the beneficial effects of the operation. Thus, Cameron, of Huddersfield, thinks that the curative action is due to the removal of the ptomaines which accumulate in the ascitic fluid and the absorption of which is responsible for the constitutional disturbance. Possibly it may be by a reduction in the activity of the inflammatory processes about the tubercles, which some have supposed are kept up and encouraged by the ascitic fluid, but it is more reasonable to suppose the existence of the latter to depend upon the activity of the former. I scarcely think we are at present in a position to give a thoroughly acceptable explanation why incision and drainage should in these cases of tubercular and other neoplasms so remarkably inhibit the growth and often induce retrograde curative changes.

Are all of these cases of cure truly tubercular? Spaeth[2] raises this question and throws doubt upon the diagnosis in the absence of the proofs afforded by the discovery of the bacillus, or the infective nature of the growths as demonstrated by inoculation. I cannot see that in practice this is an entirely forcible objection; for, as a rule, the peritoneum is the seat of miliary and nodular growths in only two affections, cancer and tubercle—the former a very rare, the latter a very common occurrence, and without histological examination, it may be impossible in certain cases to say which of the two conditions is before us.

Spaeth makes the interesting statement that there are numerous cases of other chronic diseases of the peritoneum which, at first glance, look like tuberculosis, but which on examination prove to be simply chronic peritonitis with nodular thickenings or lymphomatous growths.

[1] Quoted by Kuemmel, from Deut. med. Woch., 1880. [2] Loc. cit.

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My colleague, Dr. Welch, informs me that his experience coincides with this, and that there is a form of chronic peritonitis, usually associated with much effusion, in which the peritoneum is studded with fibroid or lymphomatous nodules. He has, in Flint's Practice (6th Edition, 1886), described these nodules as occurring in chronic serous peritonitis and speaks of the difficulty in the diagnosis between this condition and tubercular peritonitis.

The point is one not often referred to by pathologists.

Birsch-Hirschfeld[1] speaks of fine fibrous nodules occurring in large numbers on the peritoneum in chronic inflammation, similar to those which are seen upon the pleura.

Rokitansky,[2] too, speaks of papillary fibrous growths of sub-serous connective tissue as a result of hyperæmia of the peritoneum.

Prochownick,[3] in a paper upon laparotomy in chronic peritonitis, has some very interesting observations upon this subject. Two of his five cases were regarded as tuberculous until an examination was made. In the first case there were two elastic tumors by the side of the uterus. At the operation extensive union of the omentum with the pelvic peritoneum was found. There was a hemorrhagic effusion. The tumors were made up of encysted exudation. Over the intestines there was a grayish granular deposit. Although he states that this was regarded as tuberculosis, I do not see that he mentions the existence of any tubercle-like granulations or nodules. The examination of portions removed from the omentum showed that tubercles were not present, and it was evidently a case of simple peritonitis. A second case, much more remarkable, occurred in a girl of sixteen years, with a well-marked tumor in the left side of the abdomen, which upon operation was found to be composed of the coil of intestines. The entire peritoneum was covered with hundreds of small nodular tumors. On examination these were found to be made up of a lymphoid tissue, chiefly seen at the outer margin of the nodules, the centres of which had in many places undergone softening. Mundé was present at the operation in this case and refers to it (American Journal of Obstetrics, Vol. 19, page 899) as one of multiple *carcinosis* of the parietal and visceral peritoneum. The

[1] Lehrbuch der Pathologische Anatomie, 2te Auflage.
[2] 3te Auflage, Bd. 3, p. 138.
[3] Deutsche med. Wochenschrift, 1889, Number 24.

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patient, it may be mentioned, made a rapid recovery, but I see no note as to the subsequent history.

J. F. Payne[1] describes a case of minute fibrous granulations of the peritoneum associated with disseminated growths throughout the liver, possibly syphilitic, and fibrous thickening of the walls of the portal vein and bile duct. The patient, a man, aged 52, was admitted to St. Mary's Hospital under the care of Sir Edward Sieveking and died with symptoms of dropsy. The peritoneum was covered over with miliary granulations without any larger nodules and without general thickening or adhesion. There were a few litres of fluid in the cavity. Payne remarks that the peritoneal granulations were certainly not like cancer, tubercle or any generalized growth which commonly occurs in the form of a miliary eruption. They were essential fibrous outgrowths of the peritoneum and not of its serous epithelium. He says they resemble somewhat the fibrous outgrowths met with in the capsule of the liver or spleen.

I have never seen cases of this kind, unless, indeed, as is possible, I have mistaken some of these cases for fibroid tubercles. On the other hand, it is equally possible that those who have described these cases of chronic nodular peritonitis, may have confounded this condition with the healed tubercular disease. Indeed, in reference to Emma G., Case VIII, Dr. Welch tells me that had not Dr. Councilman, at the date of the operation, examined the nodules removed and demonstrated their tuberculous nature, he would, at the autopsy, which occurred four and a half months subsequently, scarcely have regarded the nodules as tubercular, so hard and fibroid had they become. In this respect the case is one of the greatest importance, as it shows how essential the examination of the nodules is, taken fresh at the time of the operation.

It must not be forgotten that in certain cases the bacilli are very difficult to find in peritoneal tuberculosis, though they may, as in the case just referred to, be most abundant even when the tubercles are very hard and fibroid. In all cases, when possible, the inoculation of a rabbit or guinea pig should supplement the histological examination.

The important practical point, however, is the relief and cure of

[1] Transactions of the Pathological Society of London, Vol. xxi.

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these cases by laparotomy, and the surgeons may well leave to the pathologist the minor question of determining the nature—whether fibroid, lymphomatous or tubercular—of the chronic peritonitis.

Among the conclusions which follow from the foregoing considerations, are :

First, that tubercular peritonitis is often a latent affection, localized in the peritoneum, which may even run its course without inducing special symptoms.

Second, that as in other local tubercular processes there is in this a natural tendency to healing, which takes place more frequently than has hitherto been supposed.

Third, that statistical evidence shows laparotomy to be in many cases a palliative, and in a certain number a curative, measure.

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A CASE OF RAYNAUD'S DISEASE ASSOCIATED WITH CONVULSIONS AND HÆMOGLOBINURIA.

By H. M. THOMAS, M. D.

The following case presents points of unusual interest, particularly in connection with the cerebral symptoms and hæmoglobinuria, which occur coincidently with the vaso-motor disturbances.

Attacks of local asphyxia of the ears, with superficial loss of substance, for three winters in succession. Local syncope of hands and feet. Epileptiform seizures, followed by hæmoglobinuria, occurring in winter.

E. W., age 26, American, car-driver by occupation, applied at the Neurological Department, September 28th, complaining of trouble in the ears and hands.

The family history was negative. He knows of no nervous disease in parents or brothers and sisters.

He was healthy and strong as a child and had the usual infantile diseases, but does not remember to have had any serious illness until the commencement of the present trouble. He does not use alcohol or tobacco to excess, and denies syphilis, but has had gonorrhœa.

The present trouble began in the winter of 1886. He had been driving his car until late at night, and on going home, into the warm room, his ears began to burn and to hurt him severely, so much so, that he was unable to sleep, and spent the night in walking about. The following day a physician told him that his ears had been frozen and ordered an external application. During the remainder of the winter the ears caused him much annoyance, becoming red and sore. During this winter he noticed, for the first time, that his hands became, as he says, "dead," and he was obliged to stop work. At this time he had his first convulsion, but whether it came on while the ears were sore and the hands affected, he does not remember. So soon as the weather became warm, these symptoms stopped, and he was able to resume work. During the summer he remained perfectly

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well, but with the onset of the cold weather, the old symptoms reappeared; the ears were swollen, blue, stiff and cold; the skin at their tips broke and they became very sore. The hands also, at times, were perfectly white, cold and numb, and from his account the sensation seems to have been impaired in them. The feet were similarly affected.

The convulsions, from which he had been entirely free during the summer, began again and became so frequent that they recurred nearly every day through the winter, and he has had as many as three a day. He says that whenever he went out in cold weather, a fit was certain to come on. The attacks always begin with a chilly feeling, as if cold water were being poured down his back; this sensation lasts long enough for him to find a place upon which to lie down; he then loses consciousness and becomes convulsed. The attack lasts about half an hour, and after it, he feels weak and has headache, the hands are tremulous, and the skin over the muscles of his calves looks blue.

Again, as summer came on, all these symptoms ceased, and he was able to return to his work. With the onset of the cold weather, in the early part of the next winter, they returned as before. He describes, in addition, a sensation in the morning, as if there were something in his stomach, and he vomits one or two mouthfuls of a greenish, bitter fluid.

After having been well the entire summer, he presents himself at the Neurological Department, alarmed at the early onset of his troubles; the ears have already given him a great deal of pain, and the morning vomiting has begun; he has had no convulsions as yet, nor have his hands or feet become white.

Present condition. Patient is a short, fairly well-nourished man, with dark eyes and hair, and a somewhat dull expression of countenance. The eye-balls are freely movable, pupils equal and react to light, on accommodation and to cutaneous irritation. An ophthalmoscopic examination revealed nothing abnormal. The muscles of the face and tongue act normally, and sensation is unimpaired. The ears are swollen and stand out prominently from the head. There is intense venous congestion of the greater portion of the helix and antehelix, and on the most prominent part of the former, there are on the right ear two white spots of necrosis; on this ear also

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there is a scooped-out loss of substance, the result of a former attack. Sensation is abolished in the deeply congested areas, but towards the meatus it is normal. To the touch the ears feel cold.

Heart sounds are normal. No abnormality noticed in the blood vessels.

The patient was treated by the daily application of the constant current to the sympathetics in the neck and also directly to the ears. The ears were kneaded between the thumbs and the fingers, and he was directed to have them rubbed at home.

On October 16th his ears had improved very much; the area of venous congestion was reduced, and he had had no pain since the first treatment. He has had no convulsions, but yesterday morning he had the sensation of a rush of blood to the head and broke into a perspiration; half an hour later his fingers became cold and stiff.

December 18th.—Patient was seen at his home. He looks pale and badly, and has quite a troublesome cough, and complains of severe pain about the epigastrium. His ears are not swollen, but on the right ear is an area of necrosis about one centimetre long. The crust has fallen from the left ear, leaving a superficial loss of substance. Sensation on the ears is normal except just in the necrotic area.

He has a great deal of pain in the ears, especially when there is much change of temperature in the air about him. This is more marked when the change is from a lower to a higher temperature. Rubbing the ears always makes them feel more comfortable.

He had a convulsion on November 28th, and one on December 16th. These began just as they did in previous winters, by the fingers becoming cold, white and dead, and the nails blue, accompanied by a good deal of pain. He had then a general sensation of chilliness, or as he said, he had a chill (and indeed, exactly similar attacks last year were supposed to be malarial). His feet became very cold, but no change of appearance was noticed in them.

These premonitory symptoms lasted for five minutes or more, when he became unconscious and was violently convulsed, requiring several men to hold him. The convulsions lasted half an hour, after which he was sleepy, and is said to have had fever.

The patient has noticed that after these attacks he has difficulty in retaining his urine, although there is no involuntary loss during

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unconsciousness, and that it is passed slowly and is always dark red and bloody looking.

Upon being closely questioned about these convulsions, it seems certain that they have only occurred since the winter in which his ears and hands first began to trouble him, and that they are always preceded by the local syncope of the fingers, and followed by the passage of bloody-looking urine.

The attacks have only occurred during winter, except that last summer he had two characteristic convulsions when the weather was cold and wet.

Attacks of local syncope of the fingers at times occur unassociated with convulsions. The fingers do not always become white and bloodless, but sometimes dark blue and even black. There has, however, never been any loss of substance.

The patient has never noticed any abnormality of vision during the premonitory stage of the attacks or at any other time.

Four microscopic slides were made of blood taken from his finger and examined carefully for the organisms of malaria, with a negative result. A fresh specimen of his urine was taken; it appeared clear and of a garnet-red color. Examination showed that it contained albumen and tube-casts, and hæmoglobin was demonstrated by the guaiacum test. There were no blood corpuscles.

January 10th.—Patient was again visited. A few days after the last visit he had had a convulsion, followed by the passage of bloody-looking urine. His ears appeared to be almost well.

A specimen of his urine was obtained, which was clear and light straw-colored, containing, however, a trace of albumen.

This case is of interest from the association of the symmetrical gangrene of the ears, with attacks of local syncope in the hands often followed by a chill, loss of consciousness and convulsions after which dark-colored urine containing hæmoglobin, albumen and tube-casts is passed.

There have been quite a number of cases of Raynaud's disease reported, in which cerebral symptoms were noticed. Thus, Cases VIII and IX, of Raynaud's series, were hysterical, and Case XIX had epileptiform attacks. In Southey's third case[1] there were attacks

[1] Transactions of the Clinical Society, Vol. XVI.

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of mania in the early part of the illness, and Southey mentioned to Barlow that he had seen another case in a young woman, the subject of epileptic dementia. Barlow notes the case of a middle-aged woman, who became the subject of delusions during a slight remission of her attacks of local asphyxia. In Weis's case[1] the patient had marked ataxic aphasia during one phase of her illness. These references occur in the appendix to Barlow's Translation of Raynaud's articles "On Local Asphyxia and Symmetrical Gangrene of the Extremities," published by the New Sydenham Society.

Morgan[2] refers also to a case reported by N. O. Bernstein,[3] in which there were epileptic fits, and one observed by Mr. Cauts, also with epileptic fits.

Bland[4] relates a case of an epileptic boy in whom the symptoms of Raynaud's disease developed after an attack of acute mania. There was also paroxysmal hæmaturia and transient impairment of vision.

Wiglesworth's[5] case in which he found, at autopsy, extensive neuritis, was in a woman suffering from epilepsy and dementia, and Palmer's[6] case was hysterical.

I have, however, been unable to find the report of any case in which convulsions were definitely associated with the occurrence of local vascular changes in the extremities.

The occurrence of intermittent hæmoglobinuria, with cases of local asphyxia and symmetrical gangrene, although not noticed by Raynaud, is still not very uncommon.[7]

[1] Wiener Klinik, 1882. [2] Lancet, Lond., July 27th, 1889, 158.

[3] Proc. of the Odessa Med. Soc., No. 12, 1884, p. 175; Abst., Lond. Med. Jour., Aug. 15th, 1885, p. 337.

[4] Lancet, Lond., 1889–1, 838. [5] Tr. Path. Soc., Lond., 1887, xxxviii, 61.

[6] Tr. Internat. Med. Cong., ix, 1887. iii, 412.

[7] See Barlow's Appendix, p. 185. Also Barlow, Ill. Med. News, Lond., 1889, iii; Cases ii, iii. Where he refers to Hutchinson (Med. Times and Gazette, 1871, ii, 678); Wilks (Med. Times and Gazette, 1879, ii, 207); Southey (St. Barth. Rpt., 1880); Dickinson (Renal and Urinary Affections, iii, 1185); Druitt (Med. Times and Gazette, April 19, 1873); Abercrombie (Arch. of Pediatrics, Oct. 1886); Myers (Trans. Clinical Soc., xviii, 336). Morgan's case (Lancet, 1889, ii, 9) and those referred to by him of Tannahill (Glasgow M. J., 1888, 624) and Beale. Bland's case is referred to above.

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ACUTE NEPHRITIS IN TYPHOID FEVER.

By WILLIAM OSLER, M. D.

Occurring early in the course of typhoid fever, nephritis and pneumonia are very apt to cause error in diagnosis, and too often the post-mortem examination gives the first intimation that there has been a general disease, masked entirely by prominent local symptoms in lungs or kidneys. There are few physicians who have not puzzled over these cases of so-called pneumo-typhoid and nephro-typhoid and have been undecided whether they had been dealing with a pneumonia or an acute nephritis with adynamic symptoms, or whether the disease has not been typhoid fever with early and unusually well marked pulmonary or renal lesions.

In my experience, the cases of typhoid fever which have set in with lobar pneumonia, and in which the symptoms of this disease have dominated the entire course, have been more common than those in which nephritis was a complication. The latter is by no means frequent, and during the ten years in which, as pathologist or physician, I was connected with the Montreal General Hospital—one of the largest fields for the study of typhoid fever on this continent—I do not remember to have seen an instance of the kind.

The renal complications which we meet with in typhoid fever may be grouped as follows: first, febrile albuminuria, usually not of much moment, even though casts be present; second, acute nephritis, often hemorrhagic, which sets in early and which gives the prominent clinical features to the case; third, a late nephritis, occurring during convalescence and which is analogous to that which follows other infectious disorders; fourth, a lymphomatous (Wagner) nephritis, also occurring late; and lastly may be mentioned, as the urinary symptoms are well marked, a post-typhoid pyelitis.

Of these, the second, acute nephritis—the *nephro-typhus* of the Germans, the *fièvre typhoïde à forme rénale* of the French—occur-

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ring at the onset or quite early in the disease, is especially likely to cause difficulty in diagnosis and forms a serious complication.

The following cases illustrate many of the clinical features of this condition.

CASE I.—*Onset with rigor. Persistent high fever. Hæmoglobinuria with albumen and tube casts. Delirium. Symptoms of perforation. Death on the 14th day of illness. Typhoid lesions in ileum; perforation. Nephritis.*

John T., aged 26, oysterman, colored, admitted to the Johns Hopkins Hospital, June 11th, 1889, complaining of pain in the lumbar and hypochondriac regions, with cough and shortness of breath. He had been ill since the 5th, when he caught a heavy cold and had pain in the back, chest and limbs, and from the description he must have had a severe rigor. Though feeling miserable he kept at work until the 10th, the day before his admission. He says that he has eaten nothing for three days.

On admission, he was complaining of lightness of the head and pain in the back. Temperature was 104°. Pulse 84, strong and full. Respirations 38, shallow. The tongue was dry and brown; the skin very hot and dry. He coughed very frequently and expectorated slightly blood-stained mucus. The abdomen was not distended; it was a little tender to the touch in the right iliac region. The lungs and heart were normal.

He was ordered antipyrin—5 grs.—and to be sponged every three hours and given a fever mixture; and, if the temperature reached 105°, to be given a graduated bath.

12th.—Passed a restless night. Temperature at 9 a. m. 102°. Cough troublesome and expectoration slightly tinged with blood. Examination of the lungs negative. The condition of the urine was as follows: Amount passed about 500 cc.; dark reddish brown in color; acid in reaction; moderate amount of greyish sediment; specific gravity 1024; albumen present. Microscopical examination showed numerous epithelial and granular casts, and much granular débris; no blood corpuscles.

Towards evening the temperature rose again to 105° and was reduced by a graduated bath.

13th.—Patient passed a restless night. Temperature between 104°

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and 105°. He had a liquid and slightly blood-stained stool. He was conscious; pulse 110, dicrotic. Tongue dry. Examination of the abdomen negative. The blood was examined on several occasions with negative result. The urine presented the same characteristics, but was perhaps a little higher in color.

14th.—Temperature at 9 o'clock 104.4°. Patient did not seem so well; was delirous; passed urine involuntarily.

15th.—Temperature again 105°, reduced by a graduated bath to 102.4°. Urine presented the same dark color; specific gravity 1022, acid in reaction; numerous tube casts, no red blood corpuscles; abundant hæmoglobin.

16th.—Temperature lower, not reaching 104°. Patient conscious; pulse 104, no longer dicrotic. Tongue swollen and moist, uniformly furred. Abdomen not distended, soft, no tenderness, a little gurgling in the right iliac fossa. Splenic dullness began at the lower border of the 7th and extended four finger's breadth vertically. Edge not palpable. Heart sounds clear. Examination of the lungs negative. 700 ounces of urine passed in the 24 hours; specific gravity 1018; color deep blood red. It contained albumen, granular and epithelial casts, hæmoglobin, but no blood corpuscles.

17th.—Temperature not above 103°. Passed a very comfortable night; was delirous at times. Pulse 120. Tongue moist and furred. Conjunctivæ a little jaundiced. Abdomen not distended. Heart sounds clear. Urine cherry red, not quite so dark but contained a larger amount of sediment; specific gravity 1016.

18th.—Patient seems better. He complained of pain in the abdomen for which he was ordered a turpentine stupe. In the evening, at 9.30, he had a rigor. The temperature fell to 97°, but after the chill it rose again to 102°. Between 8 and 11 o'clock he had four loose stools. The abdomen became very painful and a little swollen. At 3 a. m. the temperature was 104°. There was great pain in the abdomen. There was no special distension. At 7 o'clock the temperature was 104°.

19th.—At 10 a. m. the pulse was 120, and extremely feeble, scarcely to be felt. The patient seemed rational. There was no special hardness of the abdomen, no great distension. Dr. Lafleur diagnosed perforation and the propriety of a laparotomy was discussed and negatived.

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The liver dullness was almost obliterated in parasternal line. Pulmonary resonance ended at 6th rib. In 6th and 7th interspaces there was a flat tympanitic note. In mid-axillary line there was a slight liver dullness. The stomach tympany reached high to the 5th rib in the left anterior axillary line. The splenic dullness was pushed very far back. The patient became comatose and died on that day.

Post-mortem, by Dr. Welch.—In peritoneum 25 cc. of offensive brownish-yellow fluid. The lower coils of the intestines were ecchymosed in places and there was exudation of fibrin on the surface. The liver had fallen back from the abdominal wall. The diaphragm on the right side corresponded to the lower margin of the 3rd rib on the left to the upper margin of the 5th.

In thorax there were no adhesions.

In pleura there was a slight amount of blood-stained serum in each cavity. The heart weighed 325 gms.; the valves were normal, the muscular substance flabby and pale. There was general pulmonary œdema in the lungs; no foci of pneumonia.

The spleen was large and soft; and weighed 550 gms.

The capsule of the kidney was not adherent. The left weighed 226 gms. The cortex was pale-yellow. The striæ were obscured. There were no ecchymoses. The right organ was in the same condition. Both of them showed signs of decomposition.

The liver was soft and pale. The stomach presented nothing abnormal.

In the intestines the first ulcer in the ileum was 130 cm. from the valve. From this point the ulcers were numerous. Many of them were transverse, some presented the remains of shreddy, yellow sloughs. A large ulcer, 7 cm. from the valve, was irregular in shape, 6 cm. in length and 4½ cm. in breadth, with undermined edges and a sloughy, moderately congested floor. Near the centre of this were two perforations about 2 mm. apart and about 2 mm. each in diameter.

In the upper half of the large intestine there were a few scattered elevated patches with yellowish surfaces and infiltrated margins.

Microscopical examination showed in the substance of the kidney epithelium of the tubules, granular and much broken containing fatty globules of small size. There were numerous straight, thick, non-motor baccilli.

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The heart muscle showed fine granules and a few oil globules.

This case offered much difficulty in the diagnosis. We thought at first it might be malarial remittent fever but as the examination of the blood proved negative, this was excluded. The persistence of the fever at a high level and the dicrotic character of the pulse favored typhoid fever. The latter symptom was regarded as very suggestive, but it is curious that several writers have noted, particularly in these forms of renal typhoid, the absence of dicrotism. The occurrence of profuse hemoglobinuria, with his severe initial chill again suggested malaria, but we relied upon the negative character of blood examination to exclude this. There were practically no abdominal symptoms other than slight pain. It was not until the seventh day in the hospital that he had diarrhœa and this followed the chill, associated with the perforation. The rigor, the collapse temperature, the pain in abdomen with slight swelling and more particularly the almost complete obliteration of the liver dullness in the mammary line, led Dr. Lafleur to the diagnosis of perforative peritonitis.

CASE II.—*Gradual onset, with fever and cough; no rigor. Restless delirium, diarrhœa; well marked rash. Much albumen and many tube casts in urine. Remarkably low temperatures. Acute otitis media. Death about 22nd day. Extensive ulceration in ileum. Acute nephritis.*

Josie H., aged 25, was admitted to the Philadelphia Hospital on October 9th in a condition of delirium. From her friends the following history was obtained.

She had enjoyed good health with the exception of an illness of three months duration, eight years ago, which had followed the birth of a child.

She had been complaining for a few days of headache and of a feeling of fatigue and loss of appetite. She had a slight cough, but no expectoration; was feverish at times in the evening and her sleep was disturbed. She kept at work until Saturday the 5th and on Sunday the 6th took to her bed, the fever becoming more marked.

On admission, the temperature was 104.2°, pulse 123, not dicrotic, respirations 22. The patient answered questions, but rambled at times. The tongue was dry and tremulous; examination of the

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viscera was negative. There were a few bronchitic râles in the upper lobes of the lung. On the three next days the fever ranged from 102° to 103.8°. She had slight diarrhœa—two stools on the 10th, three on the 11th and three on the 12th. They were watery and yellow in color. She was very restless, constantly trying to get out of bed. The pulse ranged from 100 to 130, and on the evening of the 12th rose to 160. There was incontinuence of urine on the 10th and 11th and none was obtained for examination. The amount of urine was uncertain as it was passed involuntarily. On the 12th the patient was catheterized and six or seven oz. were withdrawn which was found to contain albumen and numerous tube casts. On the evening of the 12th the quantity had been so small that Dr. Talley, the resident physician, ordered the infusion of digitalis in half ounce doses, and a digitalis poultice on the abdomen. On the 11th rose spots were seen on the lower thoracic and epigastric regions. She continued restless and delirious, and when dozing subsultus tendinum was marked.

On the 13th she refused to take nourishment. The temperature at 3 a. m. was 100.8°; at 12 m., 101.6°; at 4 p. m., 101°; at 6.30 p. m., 97.4°. The temperature remained low all night, at 10 p. m. it registered 97.4°; at 4 a. m., 97.2°; at 7 a. m. 96.4°. The pulse was small, 115 to 120. She took nourishment better and there were 250 cc. of urine passed on the 13th.

The following notes were made of her condition at the mid-day visit:

"Face looks haggard; eyes sunken but bright; she has a distressed, frightened look; features pinched; tongue red and dry; pulse rapid and small; abdomen not distended; eruption distinct; vertical splenic dullness of four inches; urine turbid and high colored with deposit of a heavy sediment of mucus; specific gravity 1022; reaction acid; with nitric acid, a copious precipitate of albumen, one-third by bulk in settling; microscopical examination showed numerous large and coarsely granular tube casts. They were so coarse that at first they were taken for pseudo-casts of sediment. Numerous specimens were seen with the upper portions composed of mucus alone with a few particles, while the lower parts consisted entirely of these coarse granules; in the casts were also remnants of epithelium cells and, in a few, leucocytes. In the urine freshly drawn from the bladder there were

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numerous bacilli, some of which seemed to be in the tube casts. Here and there red blood corpuscles were seen but they were not numerous."

On the 14th the temperature, which had been subnormal at 7 a. m. rose by noon to 100.4°, and for the remainder of the day it kept between 104° and 105.5°. She passed 440 cc. of urine in the twenty-four hours and slept better. She also took more food.

On the 15th the temperature at 8 a. m. was 97.3° ; at 2 p. m. it rose to 101.4°, and the pulse ranged from 120 to 140. There was a discharge of blood-stained fluid from the right ear ; nothing to be seen in the meatus and no tenderness over the mastoid process. In the evening the temperature again sank below normal, and at 10 p. m., 1 a. m. and 4 a. m. it stood at 97.8°.

On the 16th the temperature was 99.2° in the morning. She had had a restless night, but looked better in the morning. She had passed urine involuntarily and the quantity for the twenty-four hours was therefore doubtful. The discharge from the right ear continued and there was no special tenderness on either side of the head.

The anomalous temperature curve and the discharge from the right ear suggested a doubt as to the true nature of the case. Puncture of spleen was made with a fine hypodermic needle and cultures prepared from the fluid. After midnight the temperature again sank.

On the morning of the 17th the temperature was 97.8°, remained about 99° all day, but at 8 p. m. sank to 97.4°. The pulse was 120, very feeble ; passed a very quiet day and seemed to recognize her friends.

On the 18th the temperature at 1 a. m. was 97° and by 8 a. m. rose to 98.8°. The urine presented the same features as before, but the granular casts were less numerous ; the amount of albumen was large, and bacilli were still seen in the urine withdrawn with careful antiseptic precautions. The continued low temperature necessitated the constant application of hot cans. The bowels had not moved for several days. The delirium persisted.

On the 19th, the temperature at 5 a. m., was 96.4° ; at 8 a. m. 96.8° ; pulse very feeble 140. She was given an injection which brought away a partly formed yellowish stool. At 2 p. m. the thermometer in the axilla could not be made to register more than 95°. The patient seemed very dull, heavy and lethargic. At 3 p. m. the temperature rose to 96.4° and by 5 p. m. it was 98.4°. She had

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hiccough through the day and more rapid respiration. The temperature at 8 p. m. was 97°; at 10 p. m. 96.4° and death took place at 3.30 on the morning of the 20th, eighteen days after taking to her bed.

Autopsy, eighteen hours after death.—The body was that of a well-made, well-nourished young woman; the skin was pale; considerable discharge from the right ear.

Abdomen. The peritoneum was smooth; the lower coils of the intestines were deeply congested and the mesentery was swollen.

Thorax. There were numerous adhesions in the right pleura; the left pleura was smooth. Pericardium was normal. The heart was of medium size and contracted, with dark clots in the right chambers; the valves were normal; muscular substance looked a little pale. Beneath the endocardium of the left ventricle there was hæmorrhage, forming a continuous layer on the septum and specially abundant about the muscular papillæ.

The lungs showed no change beyond slight hypostatic congestion at bases.

The spleen was slightly enlarged and the pulp softened; no trace on the capsule of the needle puncture.

Stomach and duodenum showed no special changes. The jejunum presented a catarrhal condition; the mucosa was thickened, and at its lower part Peyer's patches were swollen. The ileum was swollen and congested. In the upper portion there were half dozen small ulcers on the patches; the bases were formed of muscular tissue, and the edges were elevated and deeply hæmorrhagic; the lower foot of the ileum was very thick and heavy, livid in color. Three or four large large ulcers with swollen deeply hæmorrhagic edges were seen. One next to the valve was fully two inches in extent. Here and there portions of yellowish sloughs were adherent on the ulcers. The cæcum presented one or two small ulcers. The large intestine was full of yellowish-brown, liquid fæces. With the deeply hæmorrhagic character of the ulcers, it is interesting to note the absence of even a bloody tinge in the fæces.

The mesenteric glands were greatly enlarged and deeply congested. One presented a soft cheesy centre. The liver was large, the vessels full of blood, the substance rather soft.

The kidneys were enlarged and weighed together about 400 grammes; the capsules were thin and readily detached; the surface

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was pale and mottled by the presence of numerous stellated veins. On section, the substance was moist and from the larger veins blood flowed freely. There was marked contrast between the cortex and pyramids, the former being greyish-white and uniform. The tubuli Bellini appeared swollen and presented only here and there a full vessel or congested Malpighian tuft. No localized opaque areas. The pyramids were congested and the vessels and veins at the bases were distended with blood. Histologically the changes found may be thus summarized: (1) Glomerulo-nephritis involving both the vascular and capsular epithelial lining. The tuft was often partially compressed by granular débris and rounded cells which stained badly. (2) Dilatation of the convoluted tubes with extensive necrosis of the epithelium, which, as granular matter, filled the lumen. In places large tubes were distended with the pale round cells noticed in the capsules. (3) In spots atrophy of the tufts with fibroid change about them—probably an older process.

Uterus normal. Bladder tightly contracted; mucosa hyperæmic.

There was reddish, grey exudation in the right tympanum; the drum was perforated. The inflamation had not extended to the mastoid cells.

The brain showed congestion of the cortical veins; substance normal; no extension of inflammation from the ear.

This case presents an entirely different clinical picture from the first. The slow onset, fever, dry tongue, bronchitic râles, slight diarrhœa, left very little doubt at the time of her admission that the case was one of typhoid fever. The extraordinary ranges in the temperature, with the occurrence of an otitis media and the renal symptoms, shook this opinion somewhat for a few days, and it was then that Dr. Shakespeare was asked to make cultures from a blood drop from the spleen, in which were characteristic typhoid bacilli. By far the most interesting feature in this case, possibly associated with the nephritis, was the extremely irregular temperature; thus, on the 13th, the fourth day of her admission, after the temperature had been ranging from 102° to 104°, it fell at 6.30 p. m. to 97.4°, and remained about this point all night, registering at 7 a. m. 96.4°. I was afraid, at the mid-day visit, that hemorrhage or perforation had occurred. She rallied, however, and by the evening the fever was higher than it had reached previously, 105.5°. On the mornings of the 15th, 16th, 17th and

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18th, the temperature was also subnormal. It usually rose in the evening to 103° and 104°. Throughout the 19th, the thermometer only once registered 97°.

These cases, both unhappily fatal, illustrate the main features of this serious complication. Practically we have to deal with an acute, in most instances, a hemorrhagic nephritis. Naturally it adds much to the gravity of the case, and the prognosis is always rendered more dubious. Wagner,[1] however, has had five cases of recovery in succession, but the high mortality mentioned by Amat—10 deaths in 12 cases—is the more common experience.

A discussion of the relation of this form of nephritis to the typhoid poison—whether induced directly by the bacilli or by their ptomaines or the result of a mixed infection—would be beside my present purpose, which is solely clinical; nor do I think the materials are yet available for a solution of this problem, one of the most complex in the pathology of the infectious diseases.

[1] Deutsches Archiv für Klin. Med., Bds. XXV and XXXVII.

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Volume II Nos. 3-4

THE

JOHNS HOPKINS HOSPITAL REPORTS

REPORT IN GYNECOLOGY, I

By HOWARD A. KELLY, M. D.

CONTENTS.

I. The Gynecological Operating Room and the Antiseptic and Aseptic Rules in Force.
II. The Laparotomies Performed from October 16, 1889, to March 3, 1890.
III. The Report of the Autopsies in Two Cases Dying in the Gynecological Wards without Operation.
IV. Composite Temperature and Pulse Charts of Forty Cases of Abdominal Section.
V. The Management of the Drainage Tube in Abdominal Surgery.
VI. The Gonococcus in Pyosalpinx.
VII. Tuberculosis of the Fallopian Tubes and Peritoneum. Ovarian Tumor.
VIII. General Gynecological Operations from October 15, 1889, to March 4, 1890.
IX. Report of the Urinary Examination of Ninety-one Gynecological Cases.
X. Ligature of the Trunks of the Uterine and Ovarian Arteries as a Means of Checking Hemorrhage from the Uterus, etc.
XI. Carcinoma of the Cervix Uteri in the Negress.
XII. Elephantiasis of the Clitoris.
XIII. Myxo-Sarcoma of the Clitoris.
XIV. Kolpo-Ureterotomy.—Incision of the Ureter through the Vagina, for the Treatment of Ureteral Stricture.
XV. Record of Deaths following Gynecological Operations.

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THE

JOHNS HOPKINS

HOSPITAL REPORTS.

Vol. II. Nos. 3-4.

I.

THE GYNECOLOGICAL OPERATING ROOM IN THE JOHNS HOPKINS HOSPITAL, AND THE ANTISEPTIC AND ASEPTIC RULES IN FORCE.

By HOWARD A. KELLY, M. D.,
Gynecologist and Obstetrician to the Hospital.

"Show me a man's books and I will tell you of his mind," is an aphorism which might well be rendered surgically: "Show me a man's operating-room, and I will tell you of the character of his work."

Gynecology and general surgery are to-day in a transitional stage, and we see on all sides of us, men striving to attain the same ends, by means diametrically opposite. In view of this diversity it becomes important, in advance of detailed statements about cases and operations performed, and results obtained, that a surgeon managing a large clinic, and busily engaged in teaching, should at the outset say his *creed* as it were, that his readers may at once have a definite idea of the principles pervading his work.

The most intensely practical question to-day is the proper relationship between ANTISEPSIS and ASEPSIS,—to answer the questions, whether antisepsis is the father of asepsis, or whether they are distinct entities, and whether they are co-workers in the same field.

Should the surgical work throughout, from the moment the patient begins preparatory treatment for the operation, through the operation, and down through the convalescence, be a regime of antisepsis?

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Should the regime be one of asepsis, of so called "cleanliness," without the use of any germicidal drugs? Or are antisepsis and asepsis assistants one to the other, in such a way that the antisepsis may be employed to secure the asepsis, which can then be kept up for a short time, say during an operation, without any further use of antiseptics?

The decision of these important questions primarily rests not with the bacteriologist, but with the practical surgeon, who first builds up his plan of work, puts it into action, and then appeals to the bacteriologist to explain the phenomena observed. So various have been the answers to these questions that we may easily find in one and the same city, surgeons who saturate their work from beginning to end with bichloride of mercury, carbolic acid, and other antiseptic drugs, and others who simply rely upon the cleansing powers of soap and water.

The markedly individual character of the work in America to-day is due to the varying degree of enthralment of the surgeon to the idea of a regime of antiseptic drugs, or to a complete emancipation from that idea; and in either case, to the various devices adopted step by step for the purpose of carrying out the principle in view.

My own early surgical work was conducted under elaborate antiseptic precautions, using for the most part carbolic acid solutions. A careful consideration of the results obtained, both of the mortality-list in abdominal surgery, and the healing of wounds in minor gynecological surgery, convinced me that the *drug* was doing very little to bring about the good results, while it was in some instances positively harmful in its action. Its use in the peritoneal cavity was manifestly a farce, as the danger from the drug, in any germicidal strength, was greater than the danger from the germ.

These views were expressed in a paper read before the Philadelphia Obstetrical Society, May 6, 1888, entitled "Asepsis not Antisepsis: a plea for principles, not paraphernalia in Laparotomy," (American Journal Obstetrics, Vol. 19, 1886, page 1076, also Maryland Medical Journal, June 5, 1886, page 110). With this paper I published sympathetic letters from the eminent practical surgeons, Thomas Keith and Lawson Tait. My views to-day are in entire harmony with those of four years since, and while the changes in principle have been none, numerous new methods of carrying out the principle have been

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adopted; and with each change to more commodious quarters, has come a simplification and ease in the conduct of the work, which is impossible outside of specially constructed clinics.

The working rules are:

A. Antisepsis up to the moment of beginning the operation.

B. Asepsis throughout the operation.

C. Preservation of the aseptic state after the operation.

Before describing the surroundings, and the technic of the work, I will premise by stating that in a proper amplified use of the term "antisepsis" I mean *any efficient active measures taken to secure microscopic cleanliness, i. e.* freedom from germs. Used with this intent soap and water become the most valuable agents in our possession for cleansing hands and instruments, either ridding the field entirely of germs, or rendering it so clean that the action of a drug employed later will be more rapid and more effective.

The Gynecological Operating Room.

There are four essentials to a good gynecological operating room:

First, that it should be well lighted.

Second, that it should be easy to clean it thoroughly.

Third, that there should be an unlimited supply of hot and cold water.

Fourth, that instruments and all other adjuvants likely to be needed should be within instant reach.

The gynecological operating room of the Johns Hopkins Hospital is in a separate building, in the centre of the private gynecological ward, in the second story (*v.* Room 9, page 134). Clustered around it are thirteen private bed-rooms for patients, No. 5 being used as an office for physicians and nurses in charge, and No. 10 is the kitchen for the reception and preparation of the diet sent up on an elevator from the kitchen, 640 feet distant. No food is cooked in this building beyond the preparation of light sick diet on gas oven or steam table. No. 8 is the linen and general store-room. Bath room, water closets, and sink are all located in the remote part of the wing projecting from the main building, shown in the diagram. The operating room No. 9 faces the west, and is lighted by three long windows. Its floor is 16 x 20 feet, and the height of the walls 14 feet. It stands six-

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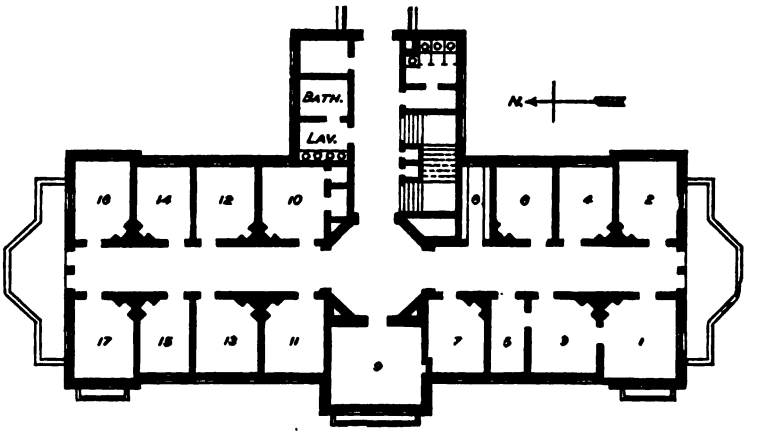
teen feet above the terrace on which the building is erected, and thirty feet above and well back from the street. The walls have a hard soap-stone finish, allowing the free use of water in cleansing, and by their light color diffusing the sunlight which enters the three western windows. All angles and corners in the whole building are rounded off (*v.* phototypes of operating room), preventing accumulations of dirt and greatly facilitating the cleansing.

The floor is laid with a light-colored, hard, encaustic German tile closely fitted, absolutely impervious to water and blood. It slopes to the discharge pipe beneath the wash basin (*v.* photos) which carries off the water used in mopping after operations. The hot and cold water



used for the preliminary washing of hands and instruments is supplied by five pairs of faucets, four over four porcelain hand-basins set in a marble slab, and two large faucets over a porcelain-lined sink, 24 x 48 in., used for washing instruments and glassware (*v.* photo). All pipes are open to view, as shown. There are no concealed nooks or corners in any part of the room. There is absolutely no "dead-space." All of the water used is either distilled or filtered. The tap water has already been filtered in the boiler vault by a Loomis filter, and yields from the cold faucet but 5–6 colonies of non-pathogenic bacteria, to the cubic centimeter, in place of 35 in the non-filtered water. The hot water thus tapped is sterile. (Dr. Abbott's report.)

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No. 1.

NORTH END OF THE OPERATING ROOM IN THE GYNECOLOGICAL DEPARTMENT.

JARS FOR BANDAGES AND TOWELS.

STEAM STERILIZER.

DRAIN FOR INSTRUMENTS AND GLASSWARE.

INSTRUMENT TABLE.

PORCELAIN BATH FOR WASHING INSTRUMENTS.

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FOOT REST EXTENDED.

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SOUTH END OF OPERATING ROOM.

No. 2. INSTRUMENT CASE.

LAPAROTOMY TABLE WITH OVARIOTOMY PAD IN PLACE AND FOOT REST EXTENDED.

CARRIAGE FOR INSTRUMENTS.

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Treatment of Instruments.

Instruments are immersed in distilled water during an operation and are never allowed to become dry afterwards until thoroughly cleaned. After an operation they are at once washed with hot water and brown soap from two to three times, and then dried very carefully and put away in the case (*v.* phototype No. 2) to the left of the fire-place.

Immediately before using the instruments at an operation, they are sterilized by steam in a newly devised Rohrbeck oven (seen in photo 1, a white chest against the rear wall to the left) at 100° C. (212° Fahrenheit), for a half-hour, when by turning a ventilator the steam is shut off, and they are automatically dried before being removed from the oven. From the oven they are at once transferred to the glass trays and basins, seen on the table in the middle of the floor in photo No. 1, and to the right in the corner in photo No. 2, in which they lie throughout the operation, immersed in hot distilled water.

The Patient.

The patient is prepared for an abdominal operation by daily baths for three or four days, or a longer period if the opportunity is afforded, and the condition of the skin requires it; in the bath the nurse carefully scrubs the skin of the abdomen, thoroughly cleaning out the umbilicus. In addition to this the patient receives twice daily a vaginal douche of 1–3000 bichloride of mercury, or a 3 per cent. solution of carbolic acid.

For abdominal hysterectomy the *technique* of vaginal cleanliness in washing out with soap and water and douching frequently, is more aggressive and thorough. The bowels are always freely opened, and the diet limited to soft, easily digested food for from three days to a week beforehand. An hour before the operation she receives a fourth of a grain of morphia and thirty grains of bismuth. When lifted on to the operating table, which is seen standing in the middle of the floor with the ovariotomy pad in place, in photo No, 2, the abdomen is again thoroughly cleansed with soap and water, and the genital hairs shaved off. This is followed by a douching with bichloride (1–1000), and again with water, to remove the bichloride. The field is then aseptic and ready for operation.

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The gauze, towels, sponges and ligatures used in all operations, are disinfected as follows:

The GAUZE is common cheese cloth, a yard wide, costing five cents per yard. It is first cut in lengths of three yards, then boiled in a solution of washing soda, two drachms to the gallon, for twenty minutes, after this it is thoroughly cleansed by washing in four distinct, hot distilled waters. Then it is wrung out and laid in a bichloride of mercury solution (1–500) for one hour, and after this transferred to alcohol, or to a 3 per cent. carbolic solution for preservation. Just before use it is lifted out of a large salt-mouth jar and wrung out and laid in hot distilled water.

The TOWELS are soaked for twenty-four hours in 1–1000 bichloride solution, and then wrung out and put in a large jar of 3 per cent. carbolic solution.

The SPONGES are bought by the pound, costing from one to two cents apiece. They are brown and gritty in the package, requiring much careful handling to render them safe for use. They are first laid in a dry cloth and pounded lightly to break up, loosen and remove the coarser grit. They are next immersed in a solution of hydrochloric acid (commercial) one drachm to one pint, and left twenty-four hours. They are taken from this and washed in from twelve to sixteen hot waters, until they no longer give the water a yellowish color; they are carefully handled all this time to separate all lumps and spicula which can be detected by eyes or fingers. They are next transferred to a 1–1000 bichloride solution for twelve hours, and from this transferred to a 3 per cent. watery solution of carbolic acid for preservation.

The LIGATURES and SUTURES are of twisted surgeon's silk, bought in skeins, sizes 1–4. They are freshly prepared every week. The skeins are opened and laid in boiling water for twenty minutes. They are next immersed in a 1–3000 bichloride solution for three hours, and transferred to alcohol for preservation, on long spools. Just before the operation the cover of the dish is removed and the necessary amount of ligature pulled out and cut off. Any ligatures left over after a laparotomy are transferred to another glass dish and used for perineal and vaginal operations.

CATGUT is bought raw from the instrument maker and prepared as follows: It is first immersed in a watery bichloride solution

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(1–1000) for twenty minutes and then transferred to absolute alcohol for one hour. It is then laid in oil of juniper wood for forty-eight hours and transferred back to alcohol for preservation.

I do not feel safe in using strands of catgut of more than 1 mm. in thickness, as we have so little control over the preliminary stages of its preparation in its extraction from the animal, and we cannot satisfactorily expose the whole thickness of heavy strands to the germ-destroying agent used.

SILK-WORM GUT is bought in bunches at from 60 to 75 cents per hundred. The curly ends are cut off and the straight pieces laid in long bottles of absolute alcohol for preservation.

Operator and Assistants.

The toilet of all persons in the room is one of the most important features. Visitors are required to put on large linen wrappers completely covering their street clothes. Nurses wear clean washed dresses and their costumes. Operator and assistants remove coat, vest, starched shirt, trowsers and shoes, donning a freshly washed, white linen suit, with short sleeves, and white canvas shoes with rubber soles. They are thus dressed in clean white clothes from head to feet. Before touching instruments, ligatures, etc., the operator and his assistants, upon entering the room in their white suits, turn to the wash basins, and there spend *at least* ten minutes washing hands, finger-nails and fore-arms with brown (oleine) soap and warm water and a moderately stiff scrubbing brush. I do not recommend the use of a metal point to clean the nails, and above all deprecate digging up the matrix. The thorough use of a good brush on short nails will do all that is necessary. After washing thoroughly in water and soap, the hands are next immersed in a saturated solution of permanganate of potash, and held there until they are uniformly deeply stained, from this they are transferred to a saturated solution of oxalic acid which removes the stain in one minute. They are then dipped in plain water, and finally laid in a bath of bichloride of mercury (1–1000) for a measured minute. The hands and arms are thus rendered clean and aseptic.

It will often happen that it is necessary after this preparation to touch parts of the patient which are not aseptic, as in arranging her

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conveniently on the table, or various articles not assuredly aseptic may be handled by accident or otherwise. After such contamination a brief, but careful, scrubbing with the soap and water, followed by a brief plunge into the bichloride solution will render the hands once more aseptic. *I attach the utmost importance to the thoroughness of the first washing*; if this is efficient, the dirt of twenty-four hours is removed and any subsequent superficial contamination, may readily be removed with less pains.

The Operation.

With such preliminary antiseptic preparations, the operation is begun under aseptic conditions. *Throughout the operation no antiseptic solutions are used.* If the intestines need washing, or the abdomen douching out, distilled water at 110° F. is used. For sponges, instruments, ligatures, and cleansing the hands from time to time, nothing but boiled distilled water is used.

The Field of Operation.

The field of operation is next shut off from the rest of the body, the table, etc., in the following manner. The patient lies on the operating table, seen in the middle of photo No. 2, which is as long as the body from the head to the knees, and the feet rest on a step 18 inches below the level of the table. The hips lie within the ovariotomy pad, which carries all the fluids used about the field of operation over the side of the table into a nine-gallon agate-ware vessel. As she is lifted on to the table the night gown is pulled well above the hips and over the elbows, so as to hold the arms up on the chest. After the preliminary shaving and disinfection, one of the prepared towels is laid across the upper part of the thighs, over a piece of rubber sheeting covering the legs. Another prepared towel is laid across the upper part of the abdomen and lower thorax. Over both of these towels and the bare abdomen between them a piece of prepared gauze is laid 66 cm. (26 in.) long, and 56 cm. (22 in.) in breadth. The gauze is slit over the linea alba from the umbilicus to pubes, the slit pulled open, and through this opening thus guarded on all sides the abdominal operation is performed.

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It is the constant duty of the operator to have a lively consciousness of what the assistants are doing with their hands, and in case of any transgression on their part to send them to the wash basin.

The Dressings.

The *dressings for the wound* in abdominal surgery are as simple as possible. If a drainage tube is used a loose plug of the sterilized gauze is dropped to the bottom, to act as a capillary drain.

The wound is thickly covered with a dry powder of iodoform and boric acid (1–7) well mixed, and this is not disturbed until the stitches are removed from the abdominal wall on the eighth day. Suppuration of the wound is rare (*v.* table in record of laparotomies, Article II). A thin layer of bichloride cotton is laid directly on the wound covered with powder, and over this a wide roll of plain absorbent cotton, 4 cm. (1½ in.) in thickness. The whole dressing is held in place by an imbricated many-tailed flannel or muslin bandage (Scultetus).

If the bandage is removed at any time during the first week fresh powder is sprinkled over the line of incision.

The nurse catheterizes the patient only if she is unable to pass her urine, first wiping off secretions from the mouth of the urethra, and then sprinkling some of the iodoform and boric acid powder within the vulvar orifice after each catheterization.

Such in brief are the simple methods of the operating room.

The full test of the method will naturally lie in the tables produced in the next article, and in the future course of the work.

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II.

THE LAPAROTOMIES PERFORMED IN THE GYNECOLOGICAL DEPARTMENT FROM OCTOBER 16, 1889, TO MARCH 3, 1890.

By HOWARD A. KELLY, M. D., and HUNTER ROBB, M. D.

The following report includes a tabulated, and an analytical record of fifty cases of abdominal section performed in the Hospital within a period of 139 days, thus averaging from the first laparotomy performed in the gynecological department, down to the last tabulated, more than one operation in every three days.

Of the *fifty* laparotomies, *thirty-two* were performed upon *white women* and *eighteen* upon *negresses*. Vitality, ability to resist shock, and rapidity of recovery seem alike in both races, and although a much more extended list of figures is needed to speak with any confidence about the relative frequency of many forms of pelvic disease, the work has thus far distinctly emphasized the statement already made, that ovarian tumors are rare in negresses and cross-breeds, while myomata are proportionately frequent. I have also seen sufficient to thoroughly convince me of the errror of the assertion, that fibroid tumors are frequent but comparatively harmless. (For a death in the case of a negress from a fibroid not operated upon, *v.* Art. III.) Gonorrhoeal salpingitis, especially the suppurative form, is frequent in the negress.

The cases are presented in the following table in the order in which the operations were performed. The diagnosis given is usually that made from the gross microscopical conditions immediately after the operation.

All tumors and specimens, without exception, have been preserved in Müller's fluid or in alcohol, for a careful microscopic study, and it is hoped that a future report dealing more particularly with the pathological problems presented will present much of interest and worthy of close study.

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ABDOMINAL

No. and Color.	Name.	Age.	Date.	Disease.	Operation.
1. W.	E. W. W.	28	10–16–89	Retroflexion of uterus.	Hysterorrhaphy by ovarian ligaments. Two silk sutures on either side around the ovarian ligaments, raising the uterus in anteflexion to the abdominal wall, where it was attached.
2. B.	M. W.	20	10–16–89	Salpingitis and ovaritis with dense adhesions. Blood cyst, right ovary (size of walnut). Tube and ovary on left side adherent and enlarged.	Extirpation of both tubes and ovaries, right tube and ovary bound down to cul-de-sac and uterus by firm adhesions which bled freely.
3. W.	M. J. E.	36	10–17–89	Myoma of uterus; large fibroid filling cavity of pelvis and pressing on ureters and producing great vesical distress.	Oöphorectomy. Both tubes and ovaries extirpated, great difficulty in delivering tubes and ovaries from beneath uterus, ligated and burnt off with cautery.
4. W.	H.	21	10–17–89	Retroflexion of uterus with large varicose veins of broad ligaments.	Hysterorrhaphy. Two silk ligatures passed around each ovarian ligament. Varicose veins tied with two silk ligatures on right and one on left side.
5. B.	M. L. S.	19	10–18–89	Salpingitis and enlarged adherent cirrhotic ovaries.	Extirpation of both tubes and ovaries. General adhesions.
6. W.	B. N.	42	10–18–89	Large multilocular ovarian cyst. Left side. Tubercular peritonitis.	Extirpation, adherent to uterus by firm adhesions, ovary size of lemon, cystic; both tubes and ovaries studded with miliary tubercles.
7. W.	A. L.	21	10–22–89	Pyosalpinx on left side. Tube and ovary firmly adherent. Right side slightly adherent.	Both tubes and ovaries extirpated.

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SECTIONS.

DRAIN.	DURATION OF OPER.	TEMP. AT ANY TIME ABV. 100°.	STITCH H. ABSCESS.	REC-OV'D.	DIED.	REMARKS.	WHOLE NO.
Yes, 24 hrs.	12 minutes.	No.	No.	R.		Uterus permanently fixed in anteflexion. Discharged 11-14-89	198
Yes.	20 minutes.	No.	No.	R.		Discharged 11-9-89	199
Yes. Iodoform gauze down to cul-de-sac.	30 minutes.	No.	No.	R.		Discharged 11-22-89 June 1890, entirely relieved.	200
No.	20 minutes.	No.	No.	R.		Uterus in perfect position, no pain. Discharged 11-16-89	201
Yes, 32 hrs.	6 minutes to sewing up of abdominal wall.	100.6° evening of second day.	No.	R.		Discharged 11-13-89	202
Yes, 60 hrs.	About 15 minutes.	No.	No.	R.		Discharged 1-14-90	203
Yes, 36 hrs.		101.4° (11th day), before and after normal.	No.	R.		Discharged 11-16-89	205

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
8. B.	E. R.	38	10–21–89	Myoma of uterus.	Both tubes and right ovar removed. Tubes firmly a herent to large intestine an fundus of uterus. Ext. coa of descending colon torn i three places, closed by silk sutures. Left tube large, fille with grumous fluid.
9. W.	S. T. McC.	32	10–22–89	Unilocular ovarian cyst of left side.	Removal of both tubes an ovaries, left side Graaffia cyst (size of fist), right sid adherent. *Hysterorrhaphy*, by one silk suture through fun dus.
10. W.	J. S. J.	36	10–22–89	Interstitial salpingitis with firm adhesions, left ovarian tumor size of lemon.	Extirpation of both tubes and ovaries.
11. W.	J. S.	31	10–23–89	Large umbilical hernia.	Omental mass protruding through hernia cut off. Hernial ring dissected out an split, brought together by bu ried catgut and deep and su perficial silk sutures.
12. B.	A. E. H.	25	10–24–89	Salpingitis and ovaritis with adhesions.	Both ovaries and tubes re moved; adhesion slight, easily separated.
13. B.	R. N.	24	10–24–89	Sero-blood cyst on right side (size of base ball), left tube and ovary enlarged, ovary full of loculi, adherent.	Both tubes and ovaries re moved, many dense adhesions separated, pedicles on both sides cauterized.
14. W.	M. M.	20	10–25–89	Hydrosalpinx of right side. Pelvic abscess on left side.	Extirpation of both tubes an ovaries, dense and universal adhesions separated, abscess covered in by rectum.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
'es. 'wo tubes, one t upper and one t lower angle f wound; 1st eft in 2 days, d in 3 days.	40 minutes.	No.	No.	R.		Discharged 12-5-89	206
No.	15 minutes.	No.	Small am't of discharge from stitch in lower angle of wound	R.		Discharged 11-19-89	207
Yes, 24 hrs.	15 minutes.	No.	No.	R.		Discharged 11-16-89	208
No.	45 minutes.	No.	Slight discharge from lower angle of wound.	R.		Discharged 11-16-89 Well 6 months later.	209
No.	10 minutes.	No.	No.	R.		Discharged 11-16-89	210
Yes, 24 hrs.	20 minutes.	4th day 101.4°, 8th day 103°, 12th day normal. Exam. by rectum and vagina detected no infiltration.	No.	R.		Discharged 12-26-89	211
Yes, 4 days.	17 minutes.	Before operation from 101.5° -102.5° for ten days. Day of operation 98.5°. After operation from 97°-101.5°	Slight discharge from two lower stitch holes.	R.		Pulse 150 before operation. Discharged 12-31-89	212

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
15. W.	S. T. P.	52	10–28–89	Salpingitis and ovaritis (chronic).	Left tube and ovary adherent to pelvic wall, exceedingly difficult to enucleate; right side free and atrophied; both tubes and ovaries removed.
16. W.	K. W. S.	41	10–29–89	Retroflexion without adhesions.	Hysterorrhaphy. Two silk sutures on either side encircling utero-ovarian ligaments.
17. B.	M. M.	28	10–29–89	Pyosalpinx and abscess of ovary on right side. Left side pyosalpinx.	Both tubes and ovaries extirpated, many and dense adhesions broken up, pedicles cauterized.
18. W.	M. H.	26	11–4–89	Salpingitis and ovaritis, with dense adhesions.	Both tubes and ovaries extirpated, all structures difficult to differentiate, tubes and ovaries when dug out covered with filamentous adhesions.
19. B.	R. B.	43	11–4–89	Salpingitis and ovaritis, with adhesions.	Both tubes and ovaries extirpated, tubes and ovaries on right side densely adherent. left tube on ovary slightly enlarged.
20. W.	S. J. L.	48	11–6–89	Ovarian tumor (unilocular) left side; small cysts on tube and mesentery (right side).	Tumor removed weighing 12 lbs., ruptured in delivery, few adhesions. Ovary and tube on right side removed.
21. B.	M. B.	34	11–7–89	Salpingitis and ovaritis, with adhesions and blood cyst of right ovary (size of walnut).	Both tubes and ovaries extirpated, dense adhesions separated from omentum.
22. B.	A. T.	23	11–7–89	Blood cyst of right ovary, size of an egg, adherent to ovary. Salpingitis and adhesions on left side.	Extirpation of both tubes and ovaries, universal dense adhesions.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
Yes, 2 days.	15 minutes.	102° (day of op.) 101.5° 5th day, 100° 11th day, 100° 13th day, 100.5° 14th day.	Profuse discharge from lower angle of wound.	R.		Discharged 12–11–89	213
No.	20 minutes.	2d day 101°, 4th day 100.2°, 6th day 100.2°, afterwards normal.	No.	R.		Discharged 12–2–89 Uterus in easy anteflexion.	214
Yes, 3 days.	15 minutes.	101.5° 9th day, rest of time normal.	No.	R.		Discharged 12–10–89	215
No.	12 minutes.	No.	No.	R.		Discharged 11–28–89	216
No.	15 minutes.	No.	No.	R.		Discharged 12–5–89	217
Yes, 3 days.	10 minutes.	No.	No.	R.		Discharged 12–2–89	218
Yes, 3 days.	16 minutes.	No.	No.	R.		Discharged 12–5–89	219
Yes, 3 days.	20 minutes.	Temp. 100.6° on 12th day, otherwise normal.	No.	R.		Discharged 11–30–89	220

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
23. W.	E. H. W.	20	11-14-89	Pyosalpinx. Both sides and cystic ovaries.	Extirpation of both ovaries and tubes, dense adhesions to intestines and omentum, the latter removed in two places. Broad thick pedicle burned off with cautery. Tube and ovary on left side not so adherent, removed with much less difficulty.
24. W.	M. W.	31	11-20-89	Salpingitis and ovaritis. Blood cyst also of left ovary.	Extirpation of both tubes and ovaries, many and dense adhesions to intestines, uterus, also to vermiform appendix. Blood cyst ruptured in delivery.
25. W.	M. B.	34	11-20-89	Pediculated myoma.	Myomectomy.
26. W.	M. F.	22	11-25-89	Pyosalpinx.	Both tubes and ovaries extirpated. Hysterorrhaphy through fundus, general velamentous adhesion; both pedicles cauterized; free pus in tubes.
27. B.	S. R.	34	11-26-89	Salpingitis and ovaritis, and myoma size of walnut on fundus.	Both tubes and ovaries extirpated; removal of myoma; right side light adhesions; left side adhesions firmer and velamentous, some broad bands, 4 cm. in length. Pedicle of myoma transfixed, stump closed by four stitches.
28. B.	J. L.	25	11-29-89	Chronic salpingitis and ovaritis (both sides).	Both tubes and ovaries extirpated, separating many dense adhesions.
29. W.	W. L.	28	11-29-89	Sarcoma (recurring) of abdominal wall.	Removal of tumor mass, by two incisions into abdominal cavity, with portion of peritoneum (size of half dollar). Peritoneal surfaces brought together by silk ligatures.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
Yes, 2 days.	17 minutes.	Temp. 100.8° on 3d day, otherwise normal.	Abscess lower angle of wound.	R.		Discharged 12-23-89	221
Yes, 24 hrs.	16 minutes to sewing of abdominal wound.	No.	No.	R.		Discharged 12-14-89	224
Yes, 48 hrs.	15 minutes.	No.	No.	R.		Discharged 12-21-89	225
Yes, 36 hrs.	20 minutes.	16th day 100.4°, 18th day 100.2°.	No.	R.		Discharged 1-6-90	226
Yes, 5 days. Secondary hemorrhage from tube 3 P. M., 11-28, followed by an attack of vomiting.	15 minutes.	100.5° 9th day, P. M.	No.		D.	Died 12-6-89 Autopsy revealed no lesion or cause of death. *V.* Autopsy Record.	227
No.		100.8° 4th day.	No.	R.		Discharged well 2-10-90	228
No.	20 minutes.	5th day 100.6°.	No.	R.		Discharged 1-6-90	229

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
30. B.	J. E.	23	11-29-89	Ovarian cyst of left side, size of fist, tube thickened, distended with fluid 3 cm. in diam. Cystic right ovary size of walnut, tube enlarged and adherent.	Extirpation of both tubes and ovaries. Many and dense adhesions separated, both cysts ruptured on delivery, allowing several ounces of straw-colored fluid to escape in abdominal cavity.
31. W.	M. A. L.	33	12-2-89	Myoma uteri (interstitial).	Myomectomy and extirpation of right tube and ovary, removal of sessile myoma, rubber ligature passed around lower part of tumor; tumor cut away and cup-shaped surface approximated by continuous stout catgut sutures in two layers.
32. B.	S. M.	22	12-4-89	Varicose veins of broad ligament, and adhesions of ovaries to pelvic wall.	Breaking up of adhesions and ligation of ovarian veins on both sides. Small cysts in ovaries punctured, letting fluid out. Nothing removed.
33. W.	M. L. F.	23	12-13-89	Chronic salpingitis and ovaritis (both sides).	Extirpation of both tubes and ovaries, separation of velamentous adhesions. Pelvis filled with encysted peritonitis.
34. W.	M. C. H.	28	12-19-89	Multilocular ovarian cyst springing from left side.	Tumor and right tube and ovary removed. Tumor adherent over a surface of 1400 sq. cm.
35. W.	N. H.	19	12-20-89	Salpingitis and ovaritis adherent to intestines and omentum; adhesions very dense.	Extirpation of both tubes and ovaries. Left tube and ovary adherent but easily delivered. Right tube and ovary adherent to intestines, and omentum. External coat of intestine torn and sutured; vermiform appendix ligated.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
Yes, 3 days.	15 minutes.	5th day 100.6°.	No.	R.		Discharged 1-1-90	230
Yes, 2½ days.	45 minutes.	No.	No.	R.		Discharged 2-1-90	232
Yes, 2½ days.	18 minutes.	No.	No.	R.		Discharged 12-31-89	233
Yes, 2½ days.	22 minutes.	No.	No.	R.		Discharged 1-8-90	234
Yes, 3 days.	20 minutes.	101.8° 2d day, 102.7° 6th day.	No.	R.		Discharged 1-18-90	235
Yes.	55 minutes.	102° 2d day, 100.4° 3d day, 100.8° 16th day.	No.	R.		Discharged 1-18-90	236

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
36. W.	E. K. B.	33	12–30–89	Myoma uteri.	Hystero-myomectomy, tumor adherent to colon by dense adhesions, muscular coats of colon torn. Left ovary and tube dug out of dense capsule.
37. W.	M. A.	21	12–31–89	Pyosalpinx, with adherent vermiform appendix.	Extirpation of both tubes and ovaries, universally adherent, with broad band-like adhesions. Cyst of right tube and ovary ruptured on delivery. Vermiform appendix removed.
38. W.	M. C. M.	46	1–3–90	Myoma of uterus.	Hystero-myomectomy; large mass involving body of uterus and two smaller fibroids attached near cervix on right side also removed. Right tube and ovary removed; tube on left ovary left in.
39. B.	H. K.	20	1–15–90	Pyosalpinx, both sides.	Extirpation of both ovaries and tubes. Right side ruptured on delivery, tube much enlarged and thickened, dense adhesions. Left side enlarged and adherent.
40. B.	H. W.	34	1–23–90	Right side, blood cyst of ovary. Left side, interstitial salpingitis.	Extirpation of both tubes and ovaries, adherent on left side to intestines. Cyst ruptured, discharging about 60 cc. of reddish fluid. Right side, ovary small, densely adherent to uterus.
41. W.	S. H. S.	53	1–27–90	Multilocular ovarian cyst.	Removal of tumor springing from right side weighing 25 lbs., slight adhesions. Left tube and ovary also removed.
42. W.	H. T.	27	2–6–90	Ovaritis and retroflexion of uterus, with slight adhesions.	Hysterorrhaphy. Extirpation of both tubes and ovaries. Ovaries size of large walnut, slight adhesions. Uterus raised against abdominal wall and one silk suture passed through fundus.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
Yes.	75 minutes.	101.5° 3d day.	No.		D.	Died 1–2–90 1 A. M.	237
Yes, 4 days.	40 minutes.	2d day 101.6°, 5th day 101.5°, 6th day 103.5°, 7th day 102.6°, 8th day 101.4°, rest of time normal.	No stitch hole abscess, but pus from abdominal cavity, from neighborh'd of appendix vermif.	R.		Discharged 2–24–90	238
Yes, 4 days.	45 minutes.	Ran from 100° on 2d day to 103° on 5th day, when she died.	No.		D.	Died 1–7–90 P. M. demonstrated ileus with local peritonitis. *V.* P. M. record.	239
Yes, 3 days.	30 minutes.	100.5° on 4th day, otherwise normal.	No.	R.		Discharged cured 2–17–90 Gonococcus demonstrated in pus from right tube.	240
Yes, 2 days.	7 minutes.	No.	No.	R.		Discharged well 4–2–90	243
Yes.	13 minutes.	No.	No.	R.		Discharged well 2–22–90	244
No.	Both tubes and ovaries removed in 4½ minutes. Abdominal wall closed in 10 minutes.	Only on 3d day 100.4°.	No.	R.		Discharged well 4–19–90 Uterus in good position against abdominal wall. Operation for lacerated perineum, 2–28–90 Complete recovery.	245

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ABDOMINAL SECTIONS—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
43. W.	M. S.	30	2-12-90	Salpingitis and ovaritis.	Extirpation of both tubes and ovaries, stumps cauterized, general adhesions, ovaries enveloped in mesentery.
44. W.	F. S.	36	2-19-90	Dermoid cyst of left ovary.	Extirpation of cyst with Fallopian tube as large as small orange. Right tube and ovary also removed. Operation closing relaxed vaginal outlet at same sitting.
45. W.	H. T.	35	2-20-90	Ovarian cyst.	Removal of cyst size of base-ball. Right tube and ovary also removed, slight adhesions on right side.
46. W.	C. R.	50	2-24-90	Multilocular ovarian cyst (right side) and omental cyst.	Removal of tumor size of foot-ball. Transfixed, tied and cut off, leaving Fallopian tube behind. Omental cyst on left side attached by a narrow margin ligated and cut off. Left ovary not found.
47. W.	A.	28	2-26-90	Oöphoritis and perioöphoritis.	Extirpation of both tubes and ovaries.
48. B.	M. H.	22	2-26-90	Cystic right ovary.	Removal of right ovary and tube. Left tube and ovary normal, not removed.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
Yes, 2 days.	10 minutes.	100.3° } even- 100.3° } ings of 2d and 3d days.	No.	R.		Discharged well 5–3–90	246
Yes.	10 minutes.	No.	No.	R.		Discharged well 4–2–90 Operation same day for lacerated perineum. Silk sutures removed on 7th and 16th days. Union perfect.	247
Yes.	15 minutes.	101° 2d day, afterwards normal.	No.	R.		Discharged well 3–28–90 All pain complained of previous to operation gone at time discharge from hospital.	248
Yes, 75 hrs.	16 minutes to completion of operation, 11 minutes more to closure of incision.	No.	No.	R.		Discharged well 4–22–90	249
Yes.	8 minutes.	101° day after operation.	No.	R.		Discharged 4–18–90 7th day became irritable and hysterical. Day and night passed urine involuntarily; later on grew violent and noisy. 6–12–90 much improved, becoming rational.	250
No.	4 minutes to suturing, 9 minutes to completion.	100.2° on 3d day, afterwards normal.	No.	R.		Discharged well 4–3–90	251

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ABDOMINAL SECTIONS.—*Continued.*

No. AND COLOR.	NAME.	AGE.	DATE.	DISEASE.	OPERATION.
49. B.	M. S.	22	2-26-90	Ovaritis and salpingitis, both sides.	Both tubes and ovaries removed, dense adhesions.
50. B.	A. T.	40	3-3-90	Myoma uteri.	Hystero-myomectomy for large fibroids. Left tube and ovary removed. Right tube and ovary not removed.

Of the *forty-two abdominal sections* for *ovarian cysts, chronic inflammatory disease of ovaries and tubes*, with and without abscess, *tuberculosis* and *hysterorrhaphy* no case died.

There are no cases of exploratory incision to record.

Of eight operations for *fibroid tumors*, two for *removal of ovaries and tubes*, three *myomectomies*, two *hystero-myomectomies*, three cases died. A careful report of the cause of death in these cases is presented in Article XV, which is a condensed statement of the autopsies made by Professors Welch and Councilman. Thirty-nine cases were drained for a period varying from twelve hours to several days.

The accompanying table is a brief analysis of the preceding compiled for ready reference, and for the determination of the frequency of certain diseases, and some of the more serious or interesting complications.

ANALYSIS OF CASES.

1. Ovarian cyst (cystoma), 6, 20, 30, 34, 41, 46.
2. Monocyst of ovary, 9, 10, 13, 45, 48.
3. Blood cyst (haematoma) of ovary, (2), (21), 22, (24), 40.
4. Dermoid cyst of ovary, 44.

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Drain.	Duration of Oper.	Temp. at any Time abv. 100°.	Stitch H. Abscess.	Rec.	Died.	Remarks.	Whole No.
es, 2 days.	15 minutes to completion.	No.	No.	R.		Discharged well 3–20–90 Letter received on April 8th, says her health is now completely restored.	252
es.	72 minutes.	100° 1st day, 100.3° 4th day, 101° 5th day, following chill 104° same night, 102.5° 7th day, afterwards normal. Temperature was distinctly caused by pneumonic trouble.	No.	R.		Flaccid abdomen throughout convalescence; perfect scar with small shallow pit at lower angle. Discharged well 4–23–90.	253

5. Ovaritis, 47.
6. Salpingitis suppurativa (pyosalpinx), 7, (17), 23, 26.
7. Salpingitis and ovaritis suppurativa (abscess of ovary and tube), 17, 37, 39.
8. Salpingitis and ovaritis with moderate adhesions, 5, 12, 15, (22), (27), (40), (42), 43, (45).
9. Salpingitis and ovaritis with dense adhesions, 2, (7), (10), (13), 18, 19, 21, 24, 28, (30), 33, 35, 49.
10. Hydrosalpinx and pelvic peritonitis, 14.
11. Tuberculosis of the Fallopian tube with tubercular peritonitis, (6).
12. Hysterorrhaphy (suspension of the malposed uterus from the abdominal wall), 1, 4, 16, and by fundus (9), (26), 42.
13. Ligature of varicose broad lig. vessels, (4), (32).
14. Puncture of small cysts of ovary and resection of ovary, 32.
15. Removal of ovaries and tubes for fibroid tumor, 3, 8.
16. Removal and exsection of fibroid tumor (myomectomy), 25, 27 (died), 31 (died).
17. Removal of fibroid tumor with part of uterus, opening the uterine cavity (hystero-myomectomy), 36, 38 (died), 50.

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18. Umbilical hernia, 4, 11.
19. Omental tumor, 46.
20. Sarcoma of abdominal wall, 29.

Some of *the more important complications* met with :—

21. Encysted peritonitis, (33).
22. Removal of vermiform appendix, (35), (37).
23. Adherent vermiform appendix separated, (24).
24. Intestinal adhesions, (23), (24), (27).
25. Intestinal suture, (8), (35), (36).
26. Omental adhesions loosened, (21).
27. Omentum partly removed, (8), (11), (23), (35).
28. Uterine adhesions, (24), 40.
29. Stitch-hole abscess, (15).
30. Slight suppuration in the lower angle of the wound, (11), (14), (23).

It will be noted that the classification used is purely clinical, and adopted for present convenience.

The figures following each name of a disease in the above list refer back to the same number of the case in the tabulated list. Thus "ovarian cyst, 6, 20," refers to cases 6 and 20 in the table, where there is an analysis of the cases with remarks. Where a case presented more than one condition, or any interesting complication, the *main pathological condition* is indicated by the figures without parentheses, while the *associated condition*, or the complication, is designated under another heading by the same figures in parentheses.

Thus it will be noticed that there were quite a number of cases of haematoma of the ovary, and in most of these the blood cysts (*not menstrual corpora nigra*) were from 2 to 3 cm., or even larger in diameter; and yet but one, No. 22, has been put down as the indication for which the operation was performed.

The others, 2, 21, 24, etc., were merely hæmatomata complicating other graver disease, and these cases will be found classified, not in parentheses, under other headings.

All the cases of OVARIAN TUMOR (CYSTOMA) were interesting, in that the operations were rapidly completed, in the absence of any

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complications, with a single exception; and the patients made quick recoveries.

There was one very bad case, No. 34, in which the tumor had grown within six months to fill out the whole abdomen, in direct ratio to the emaciation of the woman.

She had been tapped at her home, and much pressure used to drive some of the thick opalescent fluid through the trocar. This caused extravasation into the abdominal cavity, and an extensive peritonitis greatly reducing her strength. She had albumen and hyaline casts in her urine when operated upon. The cyst weighed forty pounds, its wall adhered to the abdominal wall over an area of 1400 square centimetres.

She made an excellent recovery, all albumen disappearing from the urine in the convalescence, and she is to-day in perfect health.

The monocysts, or UNILOCULAR CYSTS of the OVARY, contained a clear serous fluid. They seem to have a varying pathological significance, springing at one time from a degenerated corpus luteum, at others arising like parovarian cysts, in traces of the Wolffian bodies found in the ovarian hilum; in other cases again cysts as large as the fist arise from a single Graffian follicle. The operations in these cases were all rapid and uncomplicated and followed by rapid recoveries.

HAEMATOMATA or blood cysts of the ovary, appeared with a surprising frequency, either simply, as in the case of simple unilocular serous cysts, or complicating extensive chronic inflammatory disease of the appendages. In case 22 the cyst reached the size of a hen's egg.

The small DERMOID CYST contained fat, hair, skin, bone and cartilage. It was not adherent and was readily removed. It was of a peculiar whitish color, unlike most dermoids. Immediately after performing the abdominal section I brought the patient down to the end of the table, put the leg-holder on and sewed up a tear of the perineum extending through the sphincter.

The case of OVARITIS is at present maniacal, forming the third case of surgical mania within the writer's personal experience. Here after a rapid uncomplicated operation under chloroform narcosis, in which two elongate foetal ovaries studded with large cystic follicles were removed, the patient made a speedy recovery. On the eighth

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day the sutures were removed. The wound had healed perfectly throughout. She seemed then somewhat irritable. On the thirteenth day she was slightly hysterical and passed urine involuntarily. She talked much about her "wonderful recovery," and her freedom from pain. The note of the sixteenth day reads: "pulse 120, better than before the operation, tongue clean, appetite good, bowels regular, urine no longer involuntary, laughs at trifles, slightly fatuous." Nineteenth day: "constant state of exaltation with great flow of talk; quietly rational at times. If any one smiles in her presence she bursts out into wild, uncontrollable laughter." Twentieth day: "she is so noisy and violent, that it is necessary to tie her down in bed and put her in the isolating ward. Her average morning and evening temperature, for six days added together and divided by six, was morning 99.4° and evening 100.2.°"

My first experience of this kind was in a perineal operation, on a patient of Dr. Philip Schiedt of Philadelphia. I found out later she had at one time been an inmate of an insane asylum. Ether was given while a relaxed vaginal outlet was stitched up. She coughed from the time of the operation, and after two or three days her temperature went up to 102° F. and 104° F. She then developed a double pneumonia [septic or ether?] and suddenly in the night broke out into a wild raving mania, in which she easily overpowered three nurses who were trying to prevent her escape from the house. After two weeks of wild maniacal delirium in which it was necessary to tie her in bed and control her with large doses of morphia given hypodermically, she lay utterly prostrated, gradually recovering in several weeks more.

The second case, a patient sent me by Dr. Crilly of Philadelphia, was a young girl who had a large monocystic ovarian tumor developing within nine weeks.

She was feeble and greatly emaciated, in remarkable contrast to her swollen belly containing a tumor of over forty pounds weight. She was distressed at the time of her entrance into the hospital by the persistant assertions of some mill companions that it was a pregnancy. This persecution was continued even after the operation, and it reached her ears that a male friend was circulating the story that she had gone to the hospital for an abortion.

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I operated November 26, 1888. The tumor was emptied of a thick gluey opalescent fluid. The sac was intimately adherent to the omentum, which I had to cut off close to the transverse colon throughout its whole length. The very broad pedicle of the tumor was tied with difficulty. She was put to bed quite feeble and packed with hot water bags and stimulated. To gratify her burning thirst a few hours after the operation, she pulled up a rubber warming bottle from her feet, unscrewed the top and drank a quart of the hot water, which was immediately vomited. She also got out of bed several times.

On the third day without any apparent cause she had an exfoliative cystitis. About the fifth day the nurse reprimanded her for obstinacy in not answering questions. From this time she rapidly developed at first a talkative religious and then a violent mania, shrieking, biting and trying to escape. I noticed the same tendency in the first part of this case as in the last, to catch up the last word of any one speaking in the room, and to build up foolish sentences on it. She refused almost all nourishment. She was finally sent to the State Asylum where I saw her in March, 1889. She had then advanced phthisis, from which she finally died, never recovering her senses.

The PUS CASES all did well, in spite of the fact that they were much reduced when brought into the house, emaciated by chronic pus poisoning, and worn out with the frequent attacks of pelvic peritonitis. I pursued my usual rule in selecting the time for operation, by waiting until the acute inflammatory symptoms had subsided and the patient had rallied from the attack.

It is entirely possible that a case of this kind may be suffering when first seen, from perforation and a general suppurative peritonitis; here the operation cannot be too quickly arranged to save the patient from imminent death. There may be in this case a sudden rise in temperature followed by a drop down to or below normal, and a rapid pulse and ashy appearance plainly indicating the collapse from which it is impossible to rescue the patient except by an abdominal section and a thorough washing out and cleansing of the whole infected peritoneal sac. Such a case is now recovering in the ward of the Hospital, where several pints of fetid pus were removed from tubes and ovaries, several ounces of free pus washed out of the peritoneal sac under the colon, and the small intestines were agglutinated by a general recent peritonitis.

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These cases are the rare exceptions. The natural history of tubal and ovarian abscesses is to run a chronic course for five, ten, fifteen, and in one case upon which I operated in Philadelphia, nineteen years. The formation of numerous adhesions, by which the pus is walled off from the peritoneal cavity by repeated attacks of pelvic peritonitis, is a life-saving device and one of the most characteristic signs of the disease.

During such exacerbations the patient suffers from violent pelvic pains and lower abdominal cramps, with flushes of heat and quickened pulse.

The temperature is by no means always elevated (*v.* Art. IV). These cases are types of an acute, florid, active inflammatory process, strongly contrasting with the collapse of perforation.

CHRONIC INFLAMMATION OF THE APPENDAGES includes some fourteen of the cases, arranged under the headings 8, 9, 10, 11 (Analysis of Cases, p. 157). These were largely cases in which ovaries and tubes were bound down in the pelvis by repeatedly recurring attacks of pelvic peritonitis. The technical difficulties of the operation were often great, and at times it was almost impossible to discover the tube and ovary embedded in a mass of dense adhesions. At times they were so completely buried that the ovary appeared to lie within the folds of the broad ligament, being covered in by a smooth surface resembling peritoneum.

Drainage is the cardinal principle in all of these cases whether they bleed or not, and that by means of a glass tube, open at the lower end and perforated in its lower third with minute holes. The serous weeping from the flat adherent surfaces is sometimes profuse for twenty-four hours. The drainage tube should be inserted at the middle or lower part of the incision, about two inches above the symphysis. The tube must be long enough to hang down into the pelvis, but need not to rest upon the pelvic floor. It drains most rapidly when plugged loosely with a small extemporized gauze wick which keeps up a constant gentle capillary suction, obviating the necessity of sucking up the fluid with a syringe. The use of the tube in these cases is not simply to provide against hemorrhage, or to take up a capillary flow of blood which it has been impossible to check at the operation, but where numerous adhesions have been separated, especially of the flat

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form, and the appendages have been stripped out of an adherent bed, the tube should be inserted whether there be any hemorrhage or not, for there is often a serous weeping for from twelve to forty-eight hours from the torn adherent surfaces, sometimes to the extent of several ounces, and this always taxes the absorbing powers of the peritoneum if it is simply left to accumulate without drainage. The effort at absorption is very manifest in the temperature chart of cases not drained, as well as in the anxious expression, dry tongue, tympany, and general alarming condition, often simulating the symptoms of sepsis. When we consider further the feeble vitality of many of these subjects, the disordered state of the whole intestinal tract, as well as the great frequency of renal complications (*v.* Art. IX), it becomes very clear why we can inject salt solution into the peritoneum of a healthy dog with impunity and almost without any reaction on the system at large, and yet a small amount of fluid may sometimes severely tax the human peritoneum. Used thus for twelve or forty-eight hours, the drainage tube becomes a most efficient rapidly acting *fifth emunctory*, supplementing the kidneys and the intestinal tract during the period of their greatest depression, and disappearing as soon as its function is over, thus saving the machine a great stress of superfluous work in cleaning out débris, at a time when it is taxed to the utmost with the process of repair and recuperation.

The fact that a drainage tube is to be dropped down into the pelvis for a day or two, should make no difference in the number of sutures laid in the abdominal wall for the purpose of closing the incision. The one or two sutures in the track of the tube should be loosely tied, and not until the tube is removed drawn up and tied like the rest. In this way the track is completely closed, and the danger of leaving a weak point for a hernia is obviated.

I have adopted a new plan for the performance of HYSTERORRHAPHY, *or the correction of malpositions of the uterus by sutures.*

It has been a matter of general experience that, in spite of the best directed efforts to fasten a retroflexed uterus up in anteposition, many cases relapsed after a few months and were soon as bad as ever.

This objection is clearly due to a mechanical defect in the operation as first performed by Olshausen, Sänger and myself. By the very process of catching up the uterus by its anterior face, by means

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of the round ligaments on either side, the uterus, although raised well up and brought forwards, is slightly *feathered backwards* and the cervix thus forced to approach the vaginal outlet.

It lies thus in a position of disadvantage, as it must necessarily *hang* by its ventral attachment, and the movements of intra-abdominal pressure tend only to force the cervix nearer to the outlet.

I have obviated these difficulties *by suspending the uterus by the utero-ovarian ligaments*, reaching over the tops of the broad ligaments and catching the utero-ovarian ligaments on the *posterior* surface of the broad ligaments in two silk ligatures on either side, and suspending it by these passed into the abdominal wall on either side of the incision, low down near the symphysis.

By this procedure the uterus is brought into marked anteflexion and the cervix thrown well back.

In each case operated upon by this method, the uterus has remained in a position of natural easy anteflexion, its anterior face resting upon the anterior vaginal wall.

It would be impossible to determine by an ordinary bimanual examination that the uterus had ever been attached anywhere. The objection that hysterorrhaphy fastens the uterus up in the abdomen and thus makes an abdominal out of a pelvic organ, is not founded on fact. In every case the uterus lies within the pelvis, below a line connecting the promontory of the sacrum with the upper border of the symphysis pubis.

It must be born in mind that the anterior abdominal wall lies very close to the promontory of the sacrum, and that when the ligatures attaching the utero-ovarian ligaments to the parietal peritoneum and the sub-peritoneal tissue, are drawn tight, the flexible peritoneum and yielding adhesions sag down into the pelvis to the extent of one or two centimeters.

The operations for FIBROID TUMORS of the uterus were of three kinds.

In the first place where the tumor was comparatively small and interstitial, not larger than a three to four and a half months' pregnant uterus, choking the pelvis, and attached low down to the body of the uterus, in two cases I removed the tubes and ovaries, each time taking pains to carry the ligature under any large varicose

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vessels seen. This operation was difficult as the appendages were jammed in the pelvis and the ovaries flattened between the tumor and the pelvic walls. There is always great danger of tearing the ovary off from the hilum in these cases, causing a hemorrhage from the dilated veins, which is alarmingly abundant and difficult to control in its concealed position.

I have twice succeeded in removing the appendages in cases in which the tumor choked the pelvis, and it seemed at first impossible to get at them, by rotating the tumor with the uterus in the pelvis, like a ball in a cup, thus bringing the ovary and tube up within reach.

Both of these cases have recovered and are at present writing greatly relieved from the severe pressure symptoms.

In the second place where the fibroid tumor could be enucleated without opening the uterine cavity, either by transfixion of its pedicle, or by shelling it out of its bed (myomectomy), this method was employed rather than a supra-vaginal amputation of the uterus (hystero-myomectomy).

Of four cases, two died, while the remaining two made good recoveries. In each case the bed of the tumor was disposed of by several series of buried catgut sutures and the peritoneal surfaces carefully approximated, the pedicle dropped, the abdominal incision closed, and a drainage tube left hanging down into the pelvis. For the post mortem report *v.* Art. XV.

In the third place, of two cases of removal of a fibroid tumor with the amputation of a part of the body of the uterus by Kelly's method (*v.* American Journal of Obstetrics, Vol. XXII, No. 4, 1889), (hystero-myomectomy) one died. This patient had serious renal and hepatic disease, the extent of which it had been impossible to determine before operation. She succumbed to an ileus, with a rapidity which rendered futile any attempt at assistance by opening the abdomen and reduction, (*v.* post-mortem record, Art. XV).

The UMBILICAL HERNIA was very large, in a very fat woman. She had been through three previous abdominal sections. At the operation I first cut out all the scar tissue in the skin and then cleared the field laterally to the borders of the hernial ring, which consisted of atrophic whitish fibres of the rectus muscles, containing much cicatricial tissue. I first debated the question of splitting the

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margins of the ring and in that way getting a broad surface for union to the opposite side, but concluded it would be stronger by cutting away all flabby and cicatricial tissue. This was done, down to healthy, freely-bleeding muscular fibre, around the whole circumference of the ring. The next most important point was the suturing, which was done with silk and catgut sutures.

First, a series of silk sutures were passed, five to the inch including all the layers of the abdominal wall, just as in the case of an ordinary laparotomy where it is intended to close the abdominal wound with a single row of sutures. The sutures thus placed were left lying loose, until the peritoneum and the lower part of the rectus muscles were approximated by a deep layer of catgut sutures. Next above this was another layer also of catgut sutures bringing into apposition the recti muscles. More superficial to this layer were introduced a number of silk sutures, extending from the skin down to the recti muscles, meeting the last row. When these three rows of sutures were thus in place bringing the opposite side of the incision firmly together, then the long sutures, first introduced, were drawn up and tied.

Used in this way the long silk sutures acted by taking the tension off the walls, and relieving the short buried sutures from part of the strain of holding the walls together.

The long sutures thus acted more as splints to the opposite faces of the incision.

The short buried sutures insured the accurate apposition of rectus muscle to rectus muscle, preventing each of the long sutures from acting as the drawing string of a bag and puckering the opposite sides together in a manner insuring faulty union and a reproduction of the hernia.

Cases refused abdominal section.

These were of two classes: in the first place women came either at their own instance or sent by physicians, under the impression that pain and pelvic discomfort from which they suffered, were due to serious organic disease of the ovaries, requiring extirpation. No such disease was found and no operation was performed. In the second place two cases, one of fibroid tumor of the uterus and one of sarcoma of the ovary, entered the Hospital in such condition that I did not think either of them had a ghost of a chance to survive an operation. I refused both and they died in the Hospital (*v.* Article III).

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III.

THE REPORT OF THE AUTOPSIES IN TWO CASES DYING IN THE GYNECOLOGICAL WARDS WITHOUT OPERATION.

By HOWARD A. KELLY, M. D.

The cases included in this report entered the Hospital, private and public wards, and died without operation.

I present here a brief history of the previous condition, together with the autopsy report. Both were cases of abdominal tumors, and in neither would an operation at the time they were first seen, have offered the faintest hope of life; they had waited too long. Had they been seen at an earlier date, Case No. I would in all probability, and No. II almost certainly, have been saved.

The importance to the gynecologist of learning the exact nature of the advanced lesions associated with large pelvic tumors, is very great; and the rarity with which opportunities are offered to make a thorough anatomical investigation, induces me to make a full presentation of the facts in both of these cases.

It is here again emphasized that women having pelvic tumors should apply early for relief, also that cases will occasionally apply in which the wise man will withhold his hands.

Case No. I was a colored woman, with a large fibroid tumor, entering the Hospital January 6, 1890. She went to bed on her arrival and lay in such a state of hebetude that I could get no satisfactory history. She had a small wiry pulse, and this together with a little albumen and a few casts in the urine, her collapsed condition, and the immobility of the tumor which involved the whole uterus choking the pelvis and filling the abdomen [circumference 90 cm.] raised the suspicion of serious associated organic changes in some of the vital thoracic or abdominal viscera.

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I called Prof. Osler in consultation and he examined her, but after a careful search failed to find any tangible evidence of serious disease outside of the pelvis.

Autopsy, LXIX.

Service of Dr. Kelly. From the Pathological Laboratory Records, 2–1–1890. J. J. (Colored.)

Anatomical Diagnosis.

Myoma of uterus, with central necrosis; dilatation of ureters from pressure; pyelo-nephrosis; chronic passive congestion and emphysema of lungs; displacement of viscera and deformity of thorax, in consequence of tumor. General marasmus. Heart hypertrophy with hyaline calcific and fatty degeneration.

Exterior.

Body, 150 cm. in length; emaciated; rigor mortis present. Deep depressions beneath each clavicle, more marked on the right side; mammae atrophic; the manubrium sunken; a sharp elevation corresponding with the articulation of the second rib and sternum, more marked on the right than on the left side. Abdomen, enormously distended, a firm tumor mass felt beneath the thin abdominal walls, projecting more on the right. The right buttock, on which the weight of the body has evidently rested, is smaller than the left. The tumor is firm and smooth, except directly above the pubes, where a hard, slightly movable mass can be felt. The measurements are from the tip of the ensiform cartilage to symphysis pubis, 40 cm.; umbilicus displaced to the right, 5 cm.; greatest abdominal circumference, 79½ cm., thoracic, 69 cm. In the skin over tumor on the right side, 4 cm. below and 6 cm. to the right of the umbilicus, a round smooth cicatrix 1½ cm. in diameter; on the posterior surface of lower extremities, several large, irregular, circumscribed ecchymoses.

Subcutaneous fat slightly developed, muscles pale. Diaphragm on right side in upper portion of second intercostal space, on left, at the middle of third rib; the second intercostal space on each side 3 cm. wide, the first, 2½ cm.; the articulation of the second rib with the sternum, on the right side in the dorsal position of the body, almost directly over the articulation with the first; only 1 cm. below a perpendicular line, from the lower portion of first rib; on the left

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side this is not so pronounced. The deep cervical veins, particularly those on the right side very much dilated and engorged; right jugular, 2¼ cm. in diameter.

Peritoneal Cavity.

On opening the abdominal cavity a large smooth tumor mass appears immediately adjacent to the abdominal walls, its surface covered with large tortuous veins; firm adhesions over a small area of tumor surface, corresponding to the cicatrix in the abdominal walls, these adhesions, arranged more or less in a crescentic shape, the largest of them 6 cm. in length, easily broken through. In the middle line of the tumor mass, is a white, firm, movable nodule, and similar nodules are present on its upper left side. The extreme superior margin of the tumor corresponds to a line drawn across from the ends of the eighth ribs. On the anterior surface of the tumor, a dark-colored, flattened body, partly covered over with a thin membrane, the upper edge of which is thickened; on the right side and lower down, a similar body; closer examination shows these bodies to be enlarged and flattened ovaries, covered over in part by the broad ligaments;—the right ovary connected with the adhesions previously described; the left and both Fallopian tubes and fimbriae free from adhesions.

The veins in both broad ligaments, especially in the right, enormously distended. The growth occupies almost all of the abdominal cavity; all of the small intestines and the greater portion of the colon lie above the tumor. Peritoneum is smooth; the caecum displaced upwards, lies against the gall-bladder; the lower portion of ileum descends beneath tumor; then turns upwards to be inserted into caecum.

All of the lymphatic vessels in the lower portion of the abdomen, enormously dilated and filled with clear, slightly yellowish contents; some of the post-mesenteric vessels 5 mm. in diameter; large, dilated lymphatics seen along the ureters and over the capsule of kidney. Lymphatic glands, small.

Thoracic Cavity.

The lower border of liver corresponds to tip of ensiform cartilage in median line, elsewhere to lower border of the fifth rib; the lower

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border of descending innominate vein is ½ cm. above the sterno-clavicular articulations. Right lung is bound down by tolerably firm adhesions; left, free, and both pleural cavities, dry.

Heart.

Both layers of pericardium smooth; in pericardial cavity a few cc. of clear fluid.

In both sides of heart, fluid blood and fresh coagula; left ventricle slightly contracted; three fingers pass through valve into right ventricle; on left side only the tips of three fingers enter the valves; heart's flesh tolerably firm, of a dark brownish color. In the wall of the left ventricle, in most cases just beneath the endocardium, are numerous small yellowish foci. Thickness of left ventricle, 17 mm., right 4 mm.; length of left ventricle to lower border of aortic valve 8 cm. Endocardium presents a few pale patches. Aorta just above valves 62 mm. The entrance to left coronary artery, dilated. Weight of heart 260 grams.

Lungs.

Left lung small, anterior border thin and emphysematous, surface smooth; in the bronchial artery fresh clots, veins normal; bronchi slightly dilated; mucous membrane pale. Posterior and lower portions of lung, firm and dense. Right lung: arteries contain fresh clots, veins free; bronchi throughout lung much dilated; surface of lung smooth, save for old adhesions; anterior edge, pale, emphysematous; posterior and lower portions, firm and dense in structure,—congested.

Liver.

Gall duct open; liver, small 25 x 14 x 6½ cm., surface mottled; on section mottled, with numerous very pale areas,—otherwise, smooth, and congested. Gall bladder contains about 25 cc. clear transparent yellowish bile. Weight, 1520 grams.

Spleen.

Spleen measures 10 x 7 x 3½ cm.; capsule smooth; on section, firm, dark brownish color; trabeculae visible. Weight, 110 grams.

Stomach, Intestines, Pancreas, Supra-renal Capsules.—Normal.

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Kidneys.

Left kidney, 17 x 7 x 5½, pelvis dilated. (Pelvis of both kidneys turned to front.) Capsule adherent, surface irregular and lobulated; beneath capsule numerous whitish foci; on section throughout kidney numerous purulent foci, following lines running from pelvis towards cortex; the pyramids flattened, pelvis generally smooth, here and there covered with a fibrinous purulent exudation. Ureters, dilated, tightly adherent to the posterior surface of the tumor. The dilatation only extends from the point where the ureter separates from the tumor.

Right kidney, 17 x 6 x 4½ cm., pelvis turned to front, dilated. Kidney filled with purulent foci; in general the same condition as on the left side, the ureter is adherent to tumor for a longer distance and a longer extension of the dilated portion than on left side.

Uterus and Appendages.

The uterus is long, thin and anteflexed, extends directly over the anterior surface of tumor; is 19 cm. in length. Both Fallopian tubes patent until they reach the tumor, when they become lost in the tumor substance,—the right tube can be traced almost into uterus. Both ovaries flattened, the right, 6½ x 3½ x ½ cm., the left, 5 x 4 x ¾ cm., in the left ovary a large corpus luteum, with dark brownish clot 3 cm. in diameter.

Bladder.

Bladder, contracted, contains small amount of milky purulent urine; mucous membrane ecchymosed and of a dark red color.

Tumor.

The tumor is a large firm mass, on its posterior surface divided into two distinct lobes by deep depression corresponding to vertebrae. The small tumor on anterior aspect, which was felt previously, is found to be separated from the rest and growing in the uterus. Tumor, on section, dense, firm, its centre occupied by a large triangular cavity, longest diameter of which is 17 cm., at base, 11 cm.; this cavity is filled with clear, slightly blood-stained fluid, and with dense masses of yellowish-white, hard, elastic tissue. The greatest diameter of tumor is 25 cm. The small tumor on anterior surface, dense, firm, of somewhat opaque color.

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Microscopic Examination.

Myocardium; left ventricle, shows numerous smaller and larger opaque foci; in the centre of these small darker masses of irregular shape. Some of these patches 1–2 mm. in diameter, others very minute, the dark patches correspond to fatty degeneration of the muscle fibres and among these numerous fibres of refractive homogeneous appearance.

The fatty degeneration is chiefly in and around the foci containing hyaline fibres, but in places appears to be independent of them, and on the other hand foci of, or single, hyaline fibres occur with only few fatty fibres being observed in their neighborhood. The refractive material dissolves in glacial acetic acid rather slowly, rapidly in HCl and HNO_3, without ebullition of gas. As it dissolves, the fibres containing it swell up and lose their highly refractive appearance and appear hyaline. The refractive material is insoluble in strong caustic potash and ammonia. On the addition of H_2SO_4 the refractive substance is replaced by masses of crystals of sulphate of lime. The refractive material therefore is impregnated with a lime salt but not the carbonate. The refractive fibres are brittle and sharply contoured and break in sharp lines. None noticed in right ventricle which was examined in six different places. None found in left auricle (examined in two places). Found in all parts of left ventricle, but appear to be most abundant in upper part near left border. Stained fresh sections show a marked increase of nuclei, in and around the clumps of hyaline fibres which are themselves devoid of nuclei, and often seen broken up. The fatty, calcified foci appear to be most numerous and extensive in that part of myocardium which is near the pericardium, but they are to be found also near the endocardium. Sections placed in Flemming's solution show well the fatty degeneration which seems to be present around all calcified fibres, but these fibres themselves contain no fatty particles.

On microscopical examination the tumor is found to be a myoma.

The second case was a woman of 28 with a large sarcoma of the ovary, who entered the house vomiting, profoundly cyanosed, with a greatly distended abdomen [108 cm.] and a thready collapsing pulse of 180, and forced high costal breathing. She at no time rallied enough to give the slightest hope of a successful result from an opera-

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tion. I tapped her to discharge the excessive ascites, drawing off 5000 cc. of fluid, and she died within ten minutes, from the stasis of blood in the abdominal circulation—having bled to death into her own blood vessels.

Autopsy, LIV.

Dr. Kelly's service. From the Pathological Laboratory Records, 1–6–1890. Mrs. D., Aet. 28 years.

Anatomical Diagnosis.

Cystosarcoma of left ovary. Chronic peritonitis, ascites. Hydrothorax, compression of lungs. Passive congestion of liver, spleen and kidney.

Exterior.

Body 167 cm. in length; emaciated, pale, countenance cyanotic. Pupils dilated; posterior surface of body congested. On posterior surface of both legs, dark discolorations; on anterior surface of the left thigh is a small superficial cicatrix.

Abdomen is enormously distended, a hard firm tumor can be felt in the median line projecting anteriorly. The skin over abdomen contains numerous lineæ albicantes, these long, many over 1 cm. broad, and in these there are no glands and no hair bulbs, many of them of a silvery grey appearance and crossed by small bands of glistening cicatricial tissue. The superficial veins of the upper thorax distended. Marked oedema of the labia majora. On the anterior abdominal wall, just beneath the main projecting mass of tumor, a little to the right of the median line and 12 cm. above pubes, a small punctiform opening ½ cm. in diameter.

Peritoneal Cavity.

Subcutaneous fat slightly developed; abdominal wall exceedingly thin; muscle pale. On opening abdominal cavity, a large, irregular tumor mass appears. The anterior abdominal wall adherent over the tumor for a distance of 10x6 cm. In the abdominal cavity there is a considerable amount of fluid blood. In the median line of the tumor, just beneath the main projecting mass and opening into a cyst filled with colloid material is a fissure, extending horizontally 3½ cm.

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The tumor, which is chiefly solid, contains numerous cysts, filled with thin or fibrinous material. The omentum is thickened, congested and adheres to tumor by soft, easily-broken-down adhesions. The tumor easily lifted from its seat is attached by a broad pedicle, to the left side of the pelvis, in contact with the left corner of the uterus. The tumor removed with uterus and appendages. The left Fallopian tube extends over tumor for a distance of 15 cm., nothing apparent of left ovary. The right Fallopian tube of normal length; surface of ovary covered with cicatrices. The entire pelvic peritoneum, especially over uterus and bladder, dark red, granular, intensely congested; contains also numerous small hemorrhages.

Mucous membrane of bladder congested. Uterus is drawn to right side; its wall soft, 1½ cm. thick. Mucous membrane congested, here and there, covered with a soft, whitish material. The peritoneum, in general, reddened, the walls especially, intensely hyperaemic, with small ecchymoses, here and there. The omentum, thickened and congested. The mesenteric glands enlarged. Liver very much tilted anteriorly, extends in median line below ensiform cartilage 8 cm.; below border of ribs in mammary line, 6 cm. Liver and spleen free from adhesions. Diaphragm on right side at upper margin fifth rib, on the left in the sixth intercostal space. The pelvic peritoneum covered with a delicate, exceedingly vascular membrane, which can be stripped as a thin pellicle from the surface of peritoneum.

Intestines.

Along the border of small intestines opposite mesenteric attachment is a thin fold of tissue containing blood vessels, extending along the intestine for a distance of about one metre; the edge of this band somewhat shorter than the intestine, corresponding to attachment. Mucous membrane of intestines hyperaemic.

Thoracic Cavity.

In both pleural cavities, more especially in the left, is a large amount of clear, yellowish, fluid; on the left side, 2500 cc.; on the right, 1800 cc. Both lungs, especially the left, compressed; only one-half of the upper lobe contains any air; the balance sinks in water. Left lung has three lobes. Right lung;—portions of

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upper, middle and lower lobe contain air, remainder completely solidified. No change in lungs beyond that produced by compression. In pulmonary artery on left side a firm whitish thrombus, extending through all the main branches of the artery.

Heart.

Left ventricle, contracted; cavities on both sides of heart contain fluid blood, mixed with dark clots. Myocardium firm, dark brownish in color. Valves normal. Weight 220 grams.

Liver.

Portal vein very much enlarged, free from clots. Bile duct open. Liver is large; across lower portion of right lobe a well marked tight lace furrow. Liver measures 25 x 18 x 10 cm. Gall duct pervious. Gall bladder contains a small amount of clear yellowish bile. Surface smooth, dark in color. On section, lobules visible; marked passive congestion. Weight, 1800 grams.

Spleen.

Spleen,—12 x 6 x 3½; capsule smooth; on section, granular, trabeculae visible; Malpighian bodies apparently enlarged. Weight, 130 grams.

Supra-Renals.

Supra-renal capsules, normal.

Kidneys.

Left kidney, 13 x 6 x 3½ cm. in diameter. Capsule easily stripped off; surface somewhat mottled; pyramids congested; cortex 6 mm. markings visible. Right kidney, same size and general appearance as the left. Left, weight, 150 grams. Right, weight, 160 grams.

Pancreas.

Pancreas, normal.

Tumor.

Tumor weighs with uterus, 6507 grams. On section, almost solid; the large projecting mass in front composed of an opaque reddish

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material looking like fibrin, and in this are small cysts with clear contents, containing flakes of fibrin; in other places are smaller solid masses of greyish material, and here and there cysts with clear somewhat viscid contents; none of the smaller cysts lined with epithelium.

Sections of tumor made at various points, show its general structure to be a sarcoma, with large areas of necrosis. In places degeneration is not advanced. The tumor is almost made up of spindle and round cells; in other places the cells are more stellate, fatty and separated from one another by a loose tissue containing in its meshes much fibrin. The cysts are formed from the further degeneration of such places.

Further microscopic examination showed nothing more than brown induration of the heart and passive congestion of liver and kidney.

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IV.

COMPOSITE TEMPERATURE AND PULSE CHARTS OF FORTY CASES OF ABDOMINAL SECTION.

By HOWARD A. KELLY, M. D.

The question "What constitutes a natural temperature and pulse curve during the week following an abdominal operation?" is of the utmost practical importance.

During the week following an abdominal section every surgeon carefully scans the record of pulse and temperature, and often thus determines at a glance the well- or ill-doing of his patient, sometimes even anticipating serious changes in the general condition which first show themselves here; or again the pulse and temperature record is used as a standard to measure the gravity of other symptoms, which can be neglected as long as the pulse and temperature are low, while with an elevated pulse and temperature, they are sure signs of danger.

Some writers have insisted that the *temperature* alone is the better index of the presence or absence of inflammation going on in the peritoneal cavity, others again look more to the *pulse*, while still others insist that the *pulse and temperature* must be studied conjointly. All, however, agree on the importance of a record of both curves.

In order to discuss the temperature and pulse curves intelligently and to decide whether they are high, low or normal, we must naturally refer to some standard. This by a tacit understanding has been taken to be the pulse and temperature of health. But that such an assumption is wrong, I shall show by the following record.

To determine *the standard* with which our comparison should be made, I have prepared the following table by compounding the pulses and temperatures of twenty-six cases of abdominal section, running a smooth natural course throughout the convalescence. In all of these cases the wound healed without suppuration, there was no marked tympany, the bowels were moved at about the fourth day, and the temperature did not in any instance rise above 100.5° F. (38° C.)

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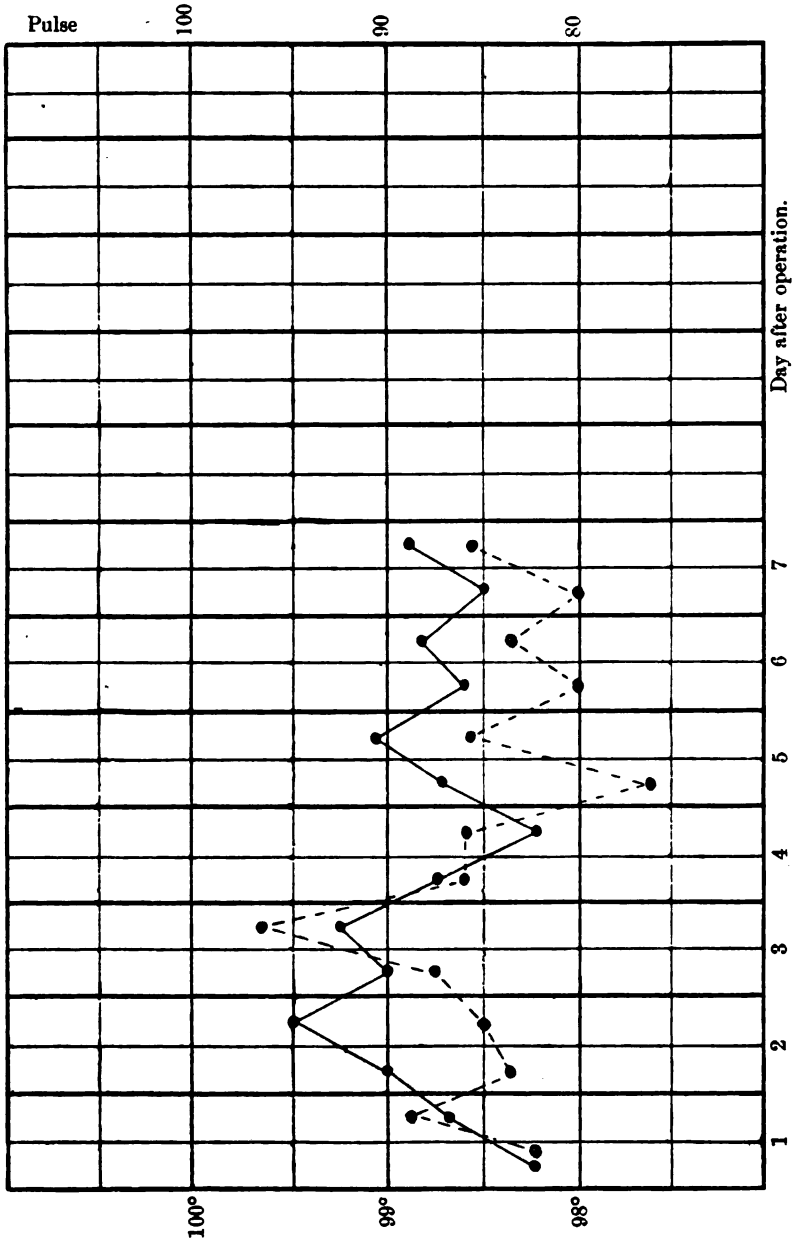

No. 1.—Composite Pulse and Temperature Chart of 26 Normal Cases of Laparotomy. The broken line is the Pulse.

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A resultant curve of all of these twenty-six cases was obtained by adding up in twenty-six rows the morning and evening temperatures, and in twenty-six other rows the pulses of all the cases, and then dividing each one of the four resultant numbers by twenty-six, when the average was found. In this average every trace of the individual peculiarity was eliminated, and thus what may be termed the composite chart representing a simple normal laparotomy was obtained.

It will be noted at once that the temperature (*v.* chart No. 1, unbroken line) is on the whole above the normal temperature of health.

Starting at 98.3° F. in the morning just before the operation it rises four-tenths of a degree by evening. On the second morning it is three-tenths of a degrec higher still, and by evening of the second day it has reached 99.5° the highest point it touched at any time. The third day witnesses a drop to 99°, and the evening rise is only to 99.3°. The fourth day is altogether exceptional, for starting with a morning temperature of 98.7°, the temperature in the evening is 98.2°, the only instance in which evening temperature is lower than morning. For the remaining three days the temperature varies slightly, between 98.5° and 99.1°.

The pulse ranges from 76 to 97, being highest on the third evening when the temperature also is highest, and lowest on the morning of the fifth day, following the greatest depression of the temperature. Twenty-one beats for the pulse, and one and three-tenths degrees Fahrenheit for the temperature, are the widest limits of excursus of these two important indicators of the progress of the patient in the average laparotomy case, doing well in all respects.

Pushing the inquiry a little further and adding up the fourteen morning and evening temperatures thus given, and then dividing by fourteen, reveals an an average bodily temperature for the whole week of 98.8°, and the same treatment of the pulse record, gives an average pulse of 85.

While thus twenty-six cases showed an average even low temperature not once reaching 100°, there were ten cases out of the fifty tabulated in Art. II presenting a chart characterized by *high* temperatures, without being abnormal in any other respect.

Dr. Farr has constructed a composite chart of these ten cases with the following result. In but three of these cases was the temperature taken for three days before operation. In these it ranged between 98.6° and 99°. The individual element is therefore not eliminated

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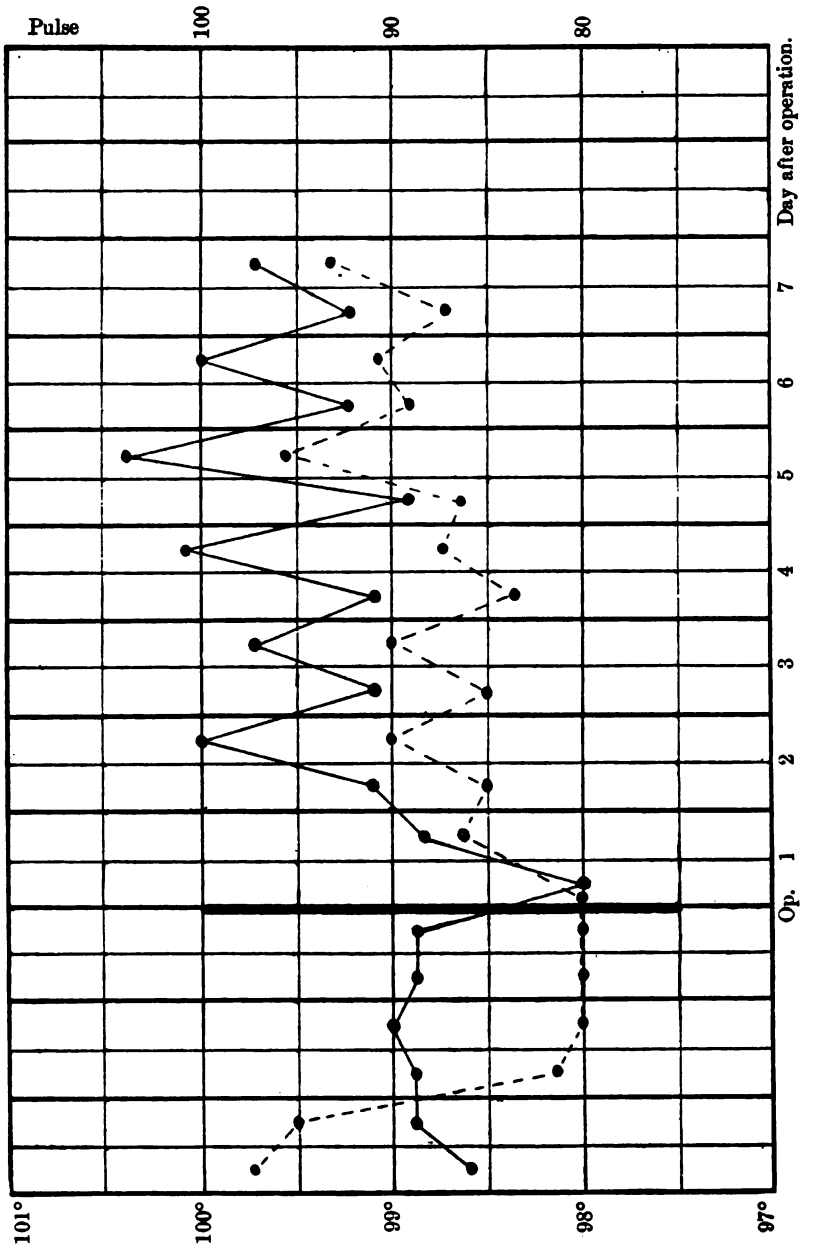

No. 2.—Composite Chart of 10 Normal Laparotomies Characterized by High Temperatures. The broken line is the Pulse.

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from this part of the chart, and it remains to be seen whether a larger number of cases will also show a greater elevation at this period.

The temperature is lowest in the morning just before the operation when it is 98°; after the operation there is a steady rise until by evening of the second day it reaches 100°, instead of 99.5°, as in chart No. 1. Almost the whole of this temperature record throughout remains *above* 99°, while in chart No. 1 it was *below* 99°. On the fourth day there is also no exception to the fact that the evening temperature is higher than the morning, as in chart No. 1. The evening temperature on this day is 100.1°. It is highest as well as lowest on the fifth day, jumping from 98.9° in the morning to 100.4° in the evening.

The pulse rate more or less closely parallels the temperature curve being lowest in the morning and highest in the evening. The greatest excursus is from 87 to 96, on the fifth day, when the temperature rises highest. The limit is from 84 to 96, a range of twelve beats, in marked contrast to the greater range of twenty-one, in the twenty-six normal cases.

This interesting comparison between these two groups of cases, one in every respect the simple natural recovery, the other normal in every respect, except in so far as it is characterized by the elevation of temperature, may be pushed further by comparing the average temperature and pulse of each for the whole week.

By adding the fourteen morning and evening temperatures and pulses of chart No. 2 in four columns, and dividing by fourteen, we get the following result: The average temperature for the whole week is 99.4°, only six-tenths higher than the normal cases, and the average pulse is 87.

There is much of positive value to be learned by these charts. In the first place we see that with present methods it is natural that there should be morning and evening variations of temperature in the simplest cases, the chart should therefore be expected to show at least a zigzag between 98.2° and 99.5°.

On the other hand about one case in four rises to 100°, and does not drop at once to stay down, but keeps rising and falling for several days, almost reaching 100.5° as late as the fifth day.

It is evident that in the absence of serious abdominal symptoms this feature of the case should never alarm the operator.

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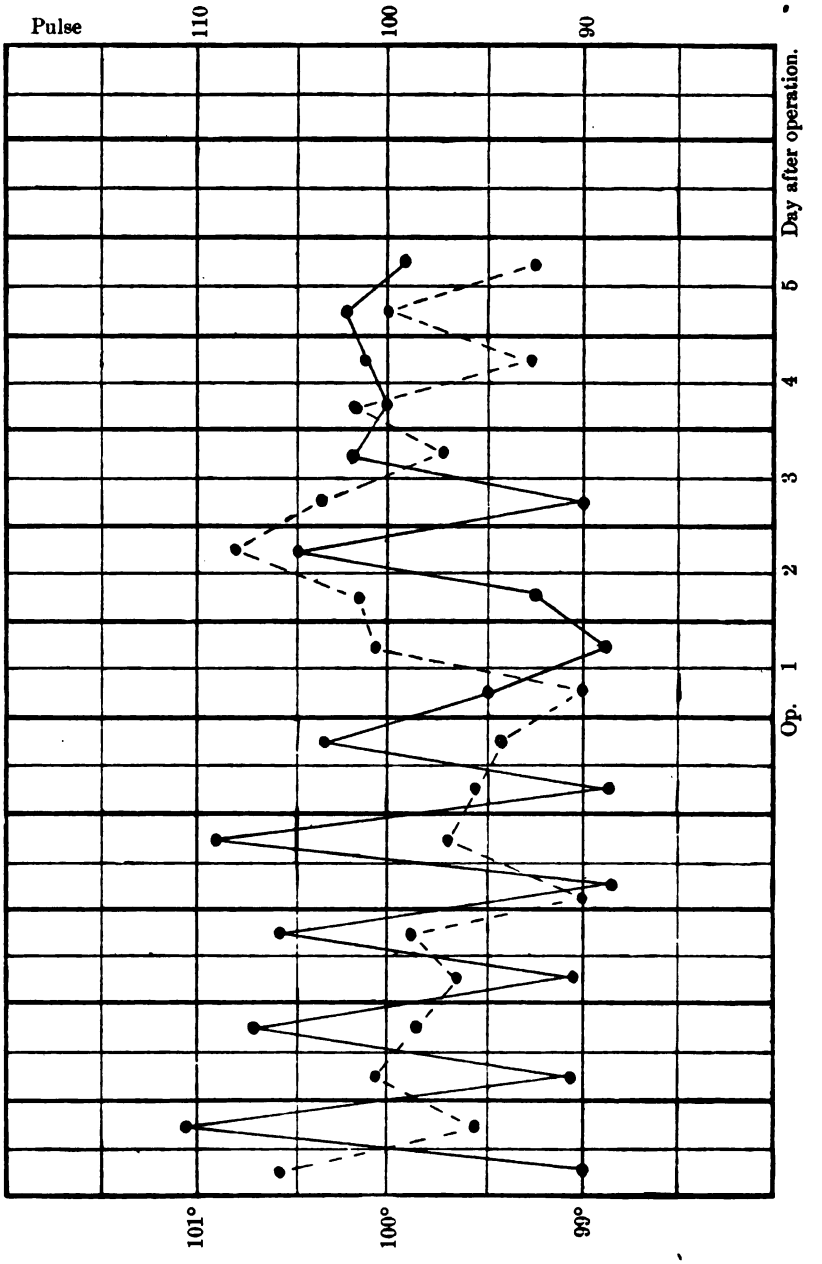

No. 3.—Composite Chart of 4 Pus Cases. The broken line is the Pulse.

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The result of similar treatment of four cases of tubal and tubo-ovarian abscess (pyosalpinx) presenting acute symptoms before operation is particularly interesting.

It will be noted in this group that the temperature taken for five days before operation shows an extraordinary uniformity of rise and fall, from 99° in the morning to 101° in the evening, the average evening temperature for the five days being 100.8°, and the average morning temperature 99°, or adding these together and dividing by two, the average bodily temperature of these cases before the operation was 99.9°.

The effect of the operation is evident in a glance at the broken curve which follows; for 24 hours the temperature drops, reaching 98.9°. On the evening of the second day it rises to 100.5°, and on the third, fourth and fifth days plays about 100°.

The average evening temperature after the operation is 99.9° and the average morning temperature 99.6°, or the medium bodily temperature 99.7°.

It is evident that the effect of the operation is not to remove all febrile disturbance at once. The bodily heat for the first few days is only reduced from 99.9° to 99.7°—two-tenths of a degree.

The most remarkable effect and one which we know practically to be of the greatest benefit, is the breaking of the hectic points on the chart. The pulse before operation averages in the morning 96.5, and 97 in the evening, while after operation it is 98.5 in the evening, and 102 in the morning.

This chart is of great value suggestively.

In the first place although but four cases are here compounded, I believe that with further investigation it will be shown to fairly represent the type of pus cases with marked febrile symptoms.

It is also demonstrated that these cases by no means assume the type of a simple laparotomy as soon as the pus is taken out. The period of defervescence is often a very slow one.

It is not my wish to attach undue importance to these three charts, based on a few cases, but to claim importance for this method of studying these cases before and after operation. A method which will obliterate the individual peculiarities of any one case, and develop those features which are characteristic of the disease as an entity, furnishes a new and valuable means of comparison of diseases among themselves.

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V.

THE MANAGEMENT OF THE DRAINAGE TUBE IN ABDOMINAL SURGERY.

By HUNTER ROBB, M. D.

A proper estimate of the advances in the *technique* of abdominal surgery, shows that the drainage tube has undoubtedly been one of the most important factors in raising it to its present status. With a still wider appreciation of its advantages, many lives which are being sacrificed yearly, would certainly be saved. Realizing therefore the paramount importance of this subject, I have thought it desirable to give a brief practical description of the management of the tube.

The use of drainage in some form is doubtless as old as the art of surgery, for Hippocrates makes mention of hollow pencils, and Galen describes leaden tubes to be used for this purpose.

In the last century, the indications for its use were extended to abdominal surgery, and Heister of Nürnberg, 1731, in his work on surgery, p. 85, recommended the use of a *wick drain* in all abdominal wounds until the discharge became perfectly healthy.

From the standpoint of to-day, however, Chassaignac of Paris, in his treatise, published in 1859, "Traité Pratique de la Suppuration et du Drainage," settles the principles of drainage upon a broad scientific basis, and is, therefore, generally accounted the discoverer of the same. Here the rubber drain first made its appearance.

With Koeberlé of Strassburg, originated the glass drainage now so universally employed in abdominal surgery.

Drainage in Pelvic Abdominal Surgery.

Drainage is to be employed to meet the following conditions:

First, to provide a means of escape for the serous oozing, which follows the separation of broad adherent surfaces.

Second, to guard against septic peritonitis from retained pus from the tube, ovary, or other viscus.

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Third, to remove the fluid in cases of persistent capillary hemorrhage.

Fourth, to provide against secondary hemorrhage in cases of hysterectomy, where the pedicle is dropped.

Fifth, to drain the peritoneal cavity, and starve out the disease in cases of chronic or tuberculous peritonitis.

Description of the Tubes.

Koeberlé's glass tubes, with trifling modifications, are still the best; (*v.* Fig. 1). These tubes are straight, of a length varying from 12 to 15 cm., and a diameter of 8, 10, and 12 mm., all lengths being made of these diameters.

Tubes curved at the end are also valuable in cases of oöphorectomy for fibro-myoma of the uterus, where it is necessary to drain Douglass' pouch over the convex surface of the tumor (*v.* Fig. 2).

Every tube should be perforated with 9–12 holes, 1 mm. in diameter, extending from the lower end, one-third the length of the tube. When the diameter of the holes is larger than 1 mm., the omentum and the small intestines are very apt to work into the lumen of the tube, forming a strangulated hernia giving rise to severe pain and vomiting with hemorrhage and sloughing.

Position and Depth of the Tube in Pelvis.

The tube should be so placed that it will drain the fluid accumulating in the most dependent portion of the pelvis. This is best accomplished by inserting the tube in Douglass' cul-de-sac, so that it lies just behind the uterus gently resting on the floor of the pelvis.

The upper portion of the tube rests in the abdominal incision, from 4 to 8 centimeters above the symphysis pubis.

Capillary Drainage through the Glass Tube, by Wick, Gauze or Cotton.

One of these substances thoroughly sterilized, and carefully placed in the tube so as to drain from the bottom, will insure a steady capillary flow of fluid from the bottom of the pelvis to the outside.

A drain made of ordinary lamp wick is the most efficient; next to this stands gauze, twisted into rolls just large enough to enter the tube easily.

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FIG. 1.

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Fig. 2.

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Other Means of Draining the Pelvis from Above.

In some cases of widespread injury to the cellular tissue of the pelvis, it is impossible to check the bleeding, and drain satisfactorily by means of the glass tube alone. Under these circumstances it is often possible to stop the hemorrhage and drain the pelvis by packing long strips of a 5 per cent. iodoform gauze, 3 cm. in width, behind the uterus and laterally, bringing them out at the lower angle of the wound. Firmer pressure can be made, and drainage secured by folding or coiling the gauze as it is placed in the pelvis, in the form of a spring, bringing one end out through a drainage tube.

The pressure on the tube, and through this on the gauze packed in the pelvis, can be regulated by tightening the abdominal binder.

A pack introduced in this way can be removed with very little disturbance by slightly raising the tube and pulling the gauze out through the tube layer by layer.

There is thus no danger of drawing out intestines or omentum with the dressing.

The Care of the Tube.

Let the tube be carefully placed, and the capillary drainage be properly provided for in the manner described, and there will be a continuous flow from the peritoneal cavity to the outside, and the tube will not need cleansing as frequently as has been thought necessary by some.

It is useless to remove the dressings every hour or two exposing the patient to the risk of a septic infection, by repeatedly cleaning out the tube. If the tube is properly arranged it can safely be left to care for itself for a period of from twelve to twenty-four hours, when it becomes necessary to expose it to remove the overlying dressings saturated by the discharge.

In fifty cases thus drained, this point has been tested by allowing the tubes to go without dressing for 24 to 48 hours, and in no case was a single unfavorable symptom observed.

The importance of perfect cleanliness in dressing the tube is not usually sufficiently appreciated. Hands, instruments, and dressings employed must be as thoroughly aseptic as at the time of operations, to avoid the danger of introducing infection from without.

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For the purpose of cleaning out the tube, the tube forceps devised by Dr. Kelly (*v.* Fig. 3) has proven very valuable in facilitating rapid, clean work.

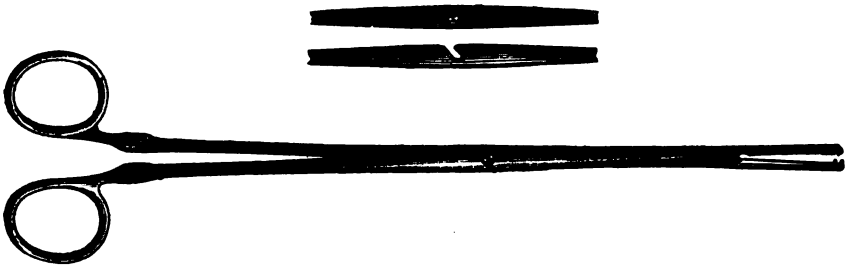
This consists of two very slender tapering-handles, crossing like scissors, and terminating below in rat teeth, to hold the little ball of cotton carried down to the bottom of the tube.

The blades are held by a new lock, devised for the purpose, as shown in the cut, above the forceps.

A piece of sterilized cotton, sponge or gauze, small enough to pass easily down to the bottom of the tube, is grasped in the forceps, and gently guided down into the pelvis, and upon withdrawal brings up the secretions.

This pledget is thrown away, and a fresh one used until the tube is dry.

FIG. 3.



This is a better method than the use of a suction apparatus, which is not efficient, and is at once contaminated and unfit for further use.

It is necessary at each dressing after cleaning the tube to rotate it, at least three times. It will sometimes be found, especially with perforations of larger calibre, that pieces of omentum as large as a split pea have become firmly fixed in the holes in the tube, forming a veritable omental hernia. Sometimes all the holes on one side will thus be choked. If gentle rotation and traction fail to effect a release, the tube must be carefully lifted up far enough to permit of a ligature being passed on the outside of the tube, around each little hernial mass in turn; after tying the ligature the tube should be cut loose by delicate, long-bladed scissors, or by a slender knife. If the intestine should be caught in this way, it must be released by traction and careful pressure from the inside of the tube, with a small piece of cotton or gauze, in the grasp of the tube forceps.

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In handling the drainage tube, the first principle is asepsis with hands and instruments, and antiseptic dressings immediately in and about the tube.

The following three modes of dressing the tube are described at length, as under each an important series of bacteriological investigations was made relative to the septic or aseptic condition of the tube contents.

The first mode, with bichloride cotton (1–200), dry or moist, and *powdered iodoform and boric acid* (1–7).

The procedure was to remove all the secretion from the tube with the bichloride cotton, held in the tube forceps, and then to dust from three to five grains of the powder mixture into the tube; then to moisten in distilled water, a piece of bichloride cotton a little smaller than the tube, and from 3 to 4 cm. longer; squeezing this fairly dry, and twisting it into a roll, it was carried to the bottom of the tube, either by inserting it like a pencil, or by pushing it with the tube forceps.

The external dressing was completed by taking a piece of the bichloride cotton about 18 cm. square, and making a hole in the centre of it, this was then laid over the tube and the bichloride cotton was piled up around it and over all until the whole surrounding area was well protected.

The second mode of dressing was with *bichloride cotton* (1–200), *and cheese cloth* sterilized and kept in alcohol, in strips from 2 to 3 cm. in width and from 14 to 18 cm. in length.

The tube was cleansed as described and dressed with one of these strips taken from the bottle, wrung out in hot, distilled water, and twisted into a long plug, which loosely filled the tube.

In some few cases, dressed in this manner, the powder mixture of iodoform and boric acid (1–7) was used.

Third mode. The dry *bichloride cotton* (1–200) was used alone, both for cleaning and plugging the tube.

Although the results of the bacteriological experiments have been practically the same, according as one or the other manner of dressing was adopted, I place the most reliance on the combination of the first and second methods, *i. e.*, the use of the bichloride cotton and sterilized gauze with powdered iodoform and boric acid, the cotton for cleaning the tube, the powder to dust down the tube, and the gauze for the drain.

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No case has ever exhibited either local or systemic toxic effect from the dressing, although it is possible that sufficient of the bichloride escapes from the dressing to inhibit the growth of any micro-organisms, which might chance to be present in the neighborhood of the tube.

When and How to Deliver the Drainage Tube.

The decision as to how long the tube shall be left in the abdomen is difficult in some cases. The general rule must be that it is safer to drain too long than for too short a time.

It must be borne in mind that the tube is inserted for the purpose of drainage, and that its function is over, and it should be removed as soon as the flow of fluid is not more than enough to wet the plug in the tube. This point may be reached in from twelve to twenty-four hours or in some instances not until the fourth or fifth day. The early removal of a tube relieves the patient of discomfort and anxiety; it also allows the fresh tissues of the tube track to collapse, promoting immediate union, and diminishing the liability to ventral hernia at a later date.

If there is but a small flow of serum on the dressing about the tube, and the general condition of the patient is good the tube may be removed without fear, and the slight accumulation left to the care of the peritoneum. If on the other hand the pulse and temperature are of such a character as to occasion any anxiety, the pulse being 120 and the temperature over 100°, although the discharge is slight it is better not to remove the tube and close the wound, until all flow has ceased.

When the tube is removed and there is a slight purulent discharge we may keep the track open by inserting a piece of twisted gauze, which is changed once in twelve or twenty-four hours, dusting in a few grains of the iodoform and boric powder at each dressing.

This allows the tube track to close up gradually, carrying off at the same time any noxious fluids which would otherwise tend to accumulate. Where there exists a free purulent discharge from the first the tube should not be removed until one or two weeks have passed, to prevent the formation of pockets of pus in the pelvis, with secondary infection.

Another method of removing the tube where the discharge is rapidly diminishing, and does not amount to more than a few drachms in

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twenty-four hours, is by rotating and raising it from one to two centimeters at each dressing. Before final removal, the tube should be cleansed as thoroughly as possible and rotated, to be sure that the intestines are free, and then placing the thumb over the end and grasping the tube between the first and middle fingers, it is slowly and gently removed. As soon as it is out, the wound is dried and the provisional suture drawn up, closing the track of the tube in the abdominal wall. The provisional sutures are one or two sutures passed through both sides of the abdominal incision in the tube track, and left loose until the tube is removed, when they are drawn up, completely closing the wound.

It is my opinion that the vast majority of abdominal cases are safer with the tube than without it. The bad results attributed to its use are usually due to carelessness of management. The care of the tube is the duty of the surgeon himself, or of some one equally conscientious, and as well instructed.

Report of Culture Experiments made with the Drainage Tubes.

These experiments were made on thirty cases in three groups, consisting of ten cases in each group.

In the first group, five of the experiments were made by stab cultures in nutrient agar-agar, two tubes being employed for each dressing. In the remaining five cases of this group, six Esmarch tubes were used at each dressing. In making the cultures, three were taken from the secretion on the end of the plug, and three from the bottom of the drainage tube. The second and third tubes were dilutions from the original inoculations in the first tube.

The results were negative, save in one case, that of a large ovarian cystoma, which was firmly adherent to the anterior abdominal wall. The inoculations made from the secretions on the end of the drainage tube, resulted in an abundant growth of *staphylococcus pyogenes aureus.* Microscopic examination of the secretions themselves revealed the presence of the ordinary pus cells. The subsequent dressings in this case gave negative results.

In the second group of inoculations, the materials used were the same as those in the second method of dressing the tube, as above described.

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Esmarch tubes were used throughout this series, six for each dressing, three being taken from the secretion on the end of the plug, and three from the secretion in the bottom of the drainage tube, after removing the plug. Six of this group were controlled by microscopic examinations. The results were all negative.

In the third group, the materials were the same as those employed with the third method of dressing the tube. None of the experiments in this series were controlled by microscopic examination. The results were also negative.

From these results one might reasonably suppose that organisms were not present in the fluid. One point, however, must be born in mind, that is the possibility of a certain amount of bichloride of mercury being dissolved out of the plug, and present, as such, in the secretions, from which the inoculations were made. In this case it is easy to see that the growth of organisms, which might have been present, would be inhibited by the bichloride taken up on the needle used in inoculating the tubes. As has been shown by recent experiments, the amount of bichloride necessary to prevent the growth of organisms which have already been exposed to its action, is very much smaller than the amount necessary to inhibit the growth of the same organisms which have not been so exposed.

Only a small proportion of these culture experiments were controlled by a microscopic examination of the fluid. The results in those cases, in which it was resorted to, however, corresponded to those obtained in the culture tubes.

A similar line of experiment, according to the improved methods recently developed for the study of organisms which have been subjected to the action of antiseptics, more particularly the bichloride of mercury, has been begun; and the results will be reported in a coming fasciculus.

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VI.

THE GONOCOCCUS IN PYOSALPINX.

By HOWARD A. KELLY, M. D.

Since the discovery by Neisser that the gonococcus occupies a position relative to gonorrhoea, similar to that held by the bacillus tuberculosis in phthisis pulmonalis, it is remarkable that pathologists and surgeons have been for the most part content to limit their demonstrations of the specific coccus to male gonorrhoea, and to florid cases of the external genital tract in the female.

The common process of argument by which pelvic inflammatory diseases, and more particularly pyosalpinx, are shown to hold a causal relationship to gonorrhoea, is usually a sequence of gratuitous suppositions, based upon the insufficient evidence of a once more or less profuse leuchorrhoea, or of an ancient gonorrhoeal attack in the husband. Oftentimes no more adequate ground is offered for the supposition, than the premises that pyosalpinx is commonly believed to be gonorrhoeal in origin, the case in hand is pyosalpinx, ergo, gonorrhoea was the cause.

In view of the grave nature of pyosalpinx, and its frequency among the lower and working classes it is very important to settle this moot point, if it were for no more practical purpose than to fix the responsibility of the origin of the attack upon a positive scientific basis.

Fresh specimens of abscesses of the tubes and ovaries should therefore always be carefully studied, and the nature of their contents noted. A few observations of such a character are at this time far more important than the record of long lists of operations where as a rule nothing appears but the facts that adhesions were dense, pus flowed, irrigation was necessary, the pelvis was drained, and the patient recovered.

It must be borne in mind however that the search for gonococci will not be an easy one. In whatever number they may have existed in

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the earlier stages of the disease, they are to be found but sparsely and often not at all in the abundant pus of the old abscess. The character of the pus and the nature of the inflammatory process throughout leave room for little doubt as to the exciting cause being a micro-organism, and the fact that repeated attempts with cultivations on nutrient agar and gelatine have yielded negative results, also lends greater probability to the supposition that the sensitive gonococcus is the organism in question.

In the case here reported it was only after two hours of patient search that Dr. Ghriskey, the gynecological assistant, at last found diplococci within the pus cells. Although so few cells were affected, each colony contained a dozen or more diplococci, characteristically grouped, pushing the nuclei of the cell a little to one side.

In the case in question this observation was made in the tube least affected, which microscopically scarcely seemed to contain any pus. No cocci at all were found in the large tube full of pus.

Smear cover-slip preparations were made after the usual method, and stained with an aqueous solution of gentian violet and mounted with xylol in Canada balsam.

Some good observers have already searched in vain. Martin[1] states that Orthmann was able to find the gonococcus in but one case of pyosalpinx, and in a letter to the writer says, "I have demonstrated the gonococcus in three cases."

Westermark[2] found the contents of a pyosalpinx to be pus and epithelial cells with undoubted gonococcus colonies.

I am not aware that it has ever yet been demonstrated in this country or England.

The following is the history of this case:

H. K., negress, single, age 20, admitted to Ward H, January 11th, 1890. Diagnosis, Pyosalpinx. Operation January 15th. Discharged February 17th, cured.

History.

Born in Baltimore; cook; well during most of her life, until she had malaria two years ago, when she was in bed for two months, and "had regular chills."

[1] Frauenkrankheiten, Wien and Leipzig, 1887, p. 380; also Ges. f. Geb. und Gyn., 28 Jan., 1887.

[2] C. f. G., 1886, p. 158, and Hygeia, 1886, Bd. 48.

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During last summer and fall she was confined to bed for a period of five months with typhoid fever and rheumatism; joints not swollen, but with pain in arms. Menses regular since 13 years of age, generally lasting from four to five days. Last period stopped on the 19th of December. Present illness began on the 21st; as she had worked in wet clothes on the 19th, she thinks she "caught cold." Pain came very suddenly while stooping down, was sharp and cutting and "doubled her up." Abdomen very tender, vomited same night and next morning, had severe headache, but no chill or fever. Bowels moved freely with medicine. Now has slight cough without expectoration.

On admission complains of severe pain in abdomen, temperature 99.2°. Is well nourished, not anaemic, skin cool and moist, tongue furred, pupils normal, heart sounds clear. Breathing partly thoracic, partly abdominal. Abdomen a little full in lower zone. Abdominal muscles a little stiff. Marked tenderness in hypogastria, and low down in iliac regions. Inguinal glands not enlarged. Temperature varies from 98° to 101° F.

She has had leuchorrhoea for about one year. Bowels constipated. Was admitted to medical wards four weeks ago, and treated for pelvic peritonitis previous to transference. Patient admits frequent coitus previous to entering an Industrial Home in 1888, and since her discharge, October 6th, 1889.

Urine catheterized. Acid. Specific gravity 1020, albumen a trace. Very slight amount of mucus. A few hyaline casts.

Examination under Ether, 1–12–90.

Vaginal outlet intact; cervix small, lying in axis of vaginal canal, curving over backwards on a tumor mass which is 8 cm. in diameter, filling up both sides of pelvic cavity, posteriorly. On left side in the broad ligament, indistinct softish mass which may be an ovary pushed down by the tumor. No structures outlined except roundish semi-fluctuant mass of tumor.

Per rectum: left ovary and tube low down in pelvis.

Diagnosis, Pyosalpinx and abscess of the ovary on right side.

Operation, 1–15–90.

Chloroform narcosis. Pulse before operation 74; after 84.

Operation, duration of fifteen minutes to beginning of the closure

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of the wound; nineteen minutes to placing last suture in the abdominal wall.

Incision, 10 cm. in length through moderately thick abdominal walls, peritoneal cavity opened in fifty seconds. Peritoneum on each side everted and caught by forceps, finger carried down into pelvis, which is found completely choked by tumors. After a careful search the fundus uteri is distinguished to the left and in front.

Enucleation commenced in a curved line, convex posteriorly, running from the uterus backwards and outwards towards the right sacro-iliac junction. After the separation of dense adhesions binding small intestines to sac, I succeeded in working my fingers down beneath the tumor and rolling it upwards and stripping it out of its bed. The cyst ruptured, with a free discharge of several ounces of pus from the incision. (Stick culture in agar-agar, and smear made at once.) Enucleation continued and large tortuous thickened tube, dilated at distal extremity with large cystic ovary, raised out of wound. Pelvic cavity now douched out with warm distilled water and whole contents carefully cleansed and all trace of pus removed before transfixing the pedicle.

The round ligament appeared in the lower angle of wound. Pedicle transfixed with silk, tied both ways and cut off; the little portion of Fallopian tube remaining on distal end of ligature, with its small stellate lumen, contained about one drop of pus. This was carefully dissected out and cut off. Surface of stump about 2 by 1 cm., white, thickened and gelatinoid. On the left side, where the disease is not so extensive as on right, more trouble in the enucleation, as it was very difficult to find the ovary and tube among masses of inflammatory thickening. Both ovary and tube markedly diseased. Elevated, transfixed and tied; burned off with Paquelin's cautery. Pelvis carefully douched until water came away almost clear. The whole posterior surface of uterus left ragged from the separation of inflammatory masses and numerous adhesions; also on right side of pelvic cavity, band-like adhesions ½ by 2 cm. binding convexity of a loop of the small intestine to the stump of the Fallopian tube at its uterine end, tied in two places and cut between. Drainage tube placed in position, incision closed by deep and superficial silk sutures. Dressing iodoform and boric acid (1–7) spread over wound, bichloride cotton over tube, absorbent cotton and Scultetus bandage.

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1–15–90. First dressing. Cotton over mouth of drainage tube and gauze plug closing it saturated with bloody discharge. Moderate amount of pain; is very fretful. Has had no anodynes. Two stick cultures and smear inoculations from drainage tube on agar-agar tubes.

1–16–90. Second dressing. Patient comfortable; pulse quiet. Abdomen perfectly flat; wound dry; very slight amount of discharge in bottom of drain. Esmarch tubes inoculated.

1–17–90. Third dressing. Doing well. Slight amount of clear fluid swabbed out of drainage tube this morning. Patient's general condition excellent. Abdomen flat. Has had no anodynes. Esmarch tubes inoculated from end of plug and bottom of drain as before.

1–18–90. No complaints. Very slight discharge; light bloody fluid removed from drainage tube, latter taken out and cotton plug (bichloride) inserted in drainage tube tract. Esmarch inoculation as before.

1–19–90. General condition very good. Wound dry. Provisional stitch in the drainage tube area tightened, bringing edges of wound in close approximation.

1–22–90. Condition excellent; abdomen flat; wound dry. All stitches removed except provisional.

2–2–90. Wound has healed without suppuration. Patient sits up out of bed to-day.

2–17–90. Discharged cured.

Temperature 100.5° on fourth day P. M., after operation. 99.5° on sixth day. Pulse highest on second day—104, otherwise temperature and pulse normal.—None of the tubes inoculated developed any growths.

Microscopic Examination.

Sections of the left Fallopian tube show the epithelium in part well preserved; the tube dilated and the dendritic condition of the mucous membrane not so marked as normal. There is some increased cellular infiltration beneath the epithelium over the entire tube. The vessels are dilated and the veins in many places contain closely-packed leucocytes. In a few places the epithelium is stripped off and there is some hemorrhage into the tissue, the cells in such places are increased in number and of a lymphoid character. Spaces are found in these

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areas with scattered remains of epithelium, probably representing the ends of deep crypts, which have been cut off from the mucous membrane by the growth of the inflammatory tissue.

The right tube shows extensive inflammation of the mucous membrane. The vessels are dilated and contain numerous leucocytes, and the epithelium is partly cast off. There is considerable hemorrhage into the sub-mucous tissue, increased accumulation of cells and small scattered islands of epithelium in the inflammatory tissue, just as in the left tube.

Smear preparations of the contents of the tubes give numerous pus and large epithelial cells, the former of the ordinary multinuclear variety, some containing micrococci arranged in pairs.

No growth took place in the culture tubes (agar-agar) inoculated with the pus, and no other bacteria were found in the pus outside of cells.

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VII.

TUBERCULOSIS OF THE FALLOPIAN TUBES AND PERITONEUM. OVARIAN TUMOR.

REMOVAL OF THE OVARIES AND FALLOPIAN TUBES, WITH DRAINAGE, FOLLOWED BY RECOVERY. DEMONSTRATION OF THE TUBERCLE BACILLUS IN SECTIONS OF THE TUBE.

By HOWARD A. KELLY, M. D.

A peculiar interest attaches to cases of tubercular infection of the peritoneum on account of the rapidity with which these patients often make a complete recovery after a simple exploratory incision with or without drainage, and with or without the removal of some principal focus of the disease, such as a tuberculous Fallopian tube.

In spite of the importance of establishing all possible parallels between tuberculosis in the peritoneum and tuberculosis in its more familiar habitat, the lungs, attempts to demonstrate the bacillus tuberculosis of the peritoneum have frequently failed, and in some of the instances in which it has been found it has been so rare as to require a prolonged patient investigation to demonstrate its presence.

Prof. Werth,[1] of Kiel, in an essay on the operative forms of genital tuberculosis, cites one case in which giant cells were found, but no tubercle bacilli could be demonstrated in the contents, or in the wall of a tuberculous tube.

In a second case tubercle bacilli were found both in the contents of the tube and in the nodules of the peritoneum.

Wiedow,[2] of Freiburg, in an article on the operative technique of genital tuberculosis, states that Prof. Maier demonstrated the presence of tubercle bacilli in a diseased tube.

[1] C. f. G., 1889, p. 499. Third Congress of the German Gyn. Soc. at Freiburg.

[2] C. f. G., 1885, p. 56. Winckel must be in error here when he states (Frauenkrankheiten, 1886, p. 585): "Tuberkelbacillen sind in den von Wiedow publicirten Fällen von Tubentuberkulose bereits durch Maier nachgewiesen worden," as Wiedow speaks of but *one case* in which they were found.

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Dr. Poten,[1] Assistant physician at the lying-in-hospital at Hanover, in describing a case of recovery from tubercular peritonitis, thus speaks of the search for bacilli.

"The demonstration of tubercle bacilli was accompanied with some difficulties: in a larger number of sections all search was in vain. I have, however, finally been able, in a series of preparations, to demonstrate sparse, but genuine tubercle bacilli. I found them without exception within the giant cells, mostly singly, occasionally two in one cell." In a case of tubercular peritonitis reported by Elliot,[2] the diagnosis was established by a microscopic examination of a nodule removed, Dr. Sears finding a single bacillus in one of the sections.

In a paper upon "Exploratory Laparotomy,"[3] by H. O. Marcy, is the following brief note: "Laparotomy showed disseminated tubercle mesenteric and over the abdominal walls. Resected a small portion for examination. The miliary masses proved to be colonies of tubercular baccilli, and cultivations were made which reproduced true." The microscopic examination was made by Dr. S. N. Nelson.

In Osler's[4] case of a girl operated on by Halsted with recovery, Councilman demonstrated the bacilli in the old quiescent pigmented nodules some months later, after death from an intercurrent disease.

In my own case the patient presented the anomaly of a tubercular peritonitis complicating ovarian cystoma, the tubercles being scattered over tubes, broad ligaments, abdominal peritoneum, and on the surface of the ovarian tumor.

Two cases in which this complication has occurred are cited in Billroth and Luecke,[5] one in the practice of Spencer Wells and the other in that of Dohrn of Königsberg.

The microscopic sections, in my case, were made by Dr. Ghriskey, who found the tubercle bacillus after a prolonged search.

History.—B. N., a patient of Dr. Barton of Plains, Pa. Irish. 42 years old. Had been married 19 years, and borne eight children, the oldest seventeen, the youngest five years old. She had one miscarriage at four months, after her sixth child, immediately following the extraction of a tooth without an anaesthetic.

[1] C. f. G., 1887, p. 35. [2] Boston Med. and Surg. Jour., May 17, 1888.
[3] Jour. Amer. Med. Assoc., Jan. 26, 1889.
[4] Johns Hopkins Hospital Reports, Vol. II, Fasciculus No. 2, p. 103.
[5] Handbuch, No. II, p. 702.

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Her last was a difficult forceps labor, completed after forty-eight hours of ineffectual pains, when she was badly torn. The perineum was operated upon three weeks later.

She has never been as well since this labor. Menstruation has come on every sixteen days, the flow being very profuse, at times even to syncope. She has spent most of her time lying on the bed.

A year ago she noticed for the first time a small lump in the lower left side of the abdomen.

This continued to grow up to the present time when a distinct mass, apparently as large as an adult's head, bulges out the left groin. She now complains much of "pain in the womb," and in the back. She "takes pleurisy every winter," and coughs constantly.

Her mother died at 40 of a pulmonary hemorrhage.

One of the children died at fifteen months with fits; the rest are living and healthy.

Examination of the urine shows no abnormality, and no tubercle bacilli can be found in the sputum.

Operation Oct. 18, 1889.—After the customary preliminary preparations [*v.* Art. II] the abdomen was opened by a 4 cm. incision, and about two pints of yellow serum escaped, coagulating on coming in contact with the air. The tumor, a multilocular ovarian cyst, of the left side, was punctured and evacuated, transfixed with silk ligatures below its base and tied off and removed, after separating a number of dense adhesions binding it to the fundus uteri, on the right side.

The right ovary was as large as a lemon and, with the tube, studded on its external surface with miliary tubercles. The visceral and parietal peritoneum and large tumor were also studded with tubercles. The small intestines were matted together in many places.

A glass drainage tube was suspended in the pelvis from the lower angle of the wound, and the incision was closed.

On the day following operation the abdomen was flat, a large amount of sero-sanguineous fluid had discharged through the tube. She vomited frequently and was therefore ordered nutrient enemata.

On the second day the discharge had ceased and the tube was removed. Her general condition was excellent.

On the nineteenth day she sat up out of bed.

Microscopic examination of the sputa again negative. Dr. Osler found no evidence of disease in the chest beyond pleuritic adhesions.

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She was discharged cured, and has since had no return of the disease.

Pathological Report.

The left ovary contains a large multilocular cyst about the size of a cocoanut with clear gelatinous contents; one of the loculi in the middle shows small papillomatous outgrowths. On the right ovary there is a single cyst, the size of a small lemon, and in this also there are small papillomatous outgrowths from the inner wall.

To each of the specimens, portions of the tubes are attached. The Fallopian tubes are three times the natural size and firmer than normal; their peritoneal covering with that of the broad ligaments is studded with very small miliary tubercles.

On the surface of the largest cyst there are numerous bands of fibrous tissue, and on these tubercles are found.

Sections of the tubes hardened in alcohol, show an intense small cell infiltration, particularly marked just beneath the epithelium, which is in part destroyed; the lumen of the tube is dilated and the small crypts of the tube are filled in part with granular detritus, pus and epithelioid cells. The small cell infiltration extends throughout the circular muscular coat, entirely destroying it. Immediately beneath the peritoneum it is less marked.

In this small cell infiltration there are innumerable tubercles, generally miliary, other larger tubercles formed by the junction of several, and diffuse tuberculous tissue; in all of these there are numerous characteristic, giant and epithelioid cells. All of the tubercles appear rather recent, in some there is no caseation, in the larger ones it is only slight. Sections of the tubes stained with carbol-fuchsine, examined for tubercle bacilli, show them to be present in small numbers in and near the giant cells. About one bacillus can be found in each section.

The more advanced and extensive tubercular disease within the tubes where caseation is just beginning, shows that the tubes formed the original nidus of the disease, from which point the infection spread to the surrounding peritoneum.

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VIII.

GENERAL GYNECOLOGICAL OPERATIONS FROM OCTOBER 15TH, 1889, TO MARCH 4TH, 1890.

By HOWARD A. KELLY, M. D.

The following tabulated list includes all of the gynecological operations performed in the Hospital, not classified under the head of abdominal sections. For the sake of convenience three cases of vaginal hysterectomy are retained in this list.

Numerically as well as practically the most important cases are the injuries of cervix and perineum. Ten operations were performed for laceration of the perineum and relaxation of the vaginal outlet. Two were for complete tear of the perineum, extending into the recto-vaginal septum. Four were double operations, both perineum and cervix being operated upon at the same sitting. In one instance a dermoid cyst of the left ovary was removed first, and following this a perineal operation was performed. There was one case of triple operation; and two of cervical operation, followed in ten days or two weeks by a perineal repair. In no case was there failure of union or faulty union. In one case there was slight suppuration.

In spite of some discredit, which has recently been cast upon the operation for laceration of the cervix uteri, I look upon it, in well selected cases, as one of the most important of our minor gynecological procedures. Where the uterus presents large, gaping, everted, lacerated lips, with much thickening and infiltration, the excision of the wedges of tissue along the borders and up into the angles of the tear, by freely depleting the cervix and emptying the numerous cysts, affords an excellent means of making a profound impression upon the heavy, congested, lower uterine segment. Besides the restoration of a diseased uterus, to a normal condition, I have seen the

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most excellent results follow this operation, in the relief of pelvic aches and dysmenorrhoea, as well as the cure of sterility.

The perineal operations were performed in three ways:

First. In cases of complete tear, symmetrical denudations were made on either side, extending forward to a point just below the level of the urethra, and back to include the sphincter ani fibres, and up into the septum well beyond the angle of the tear. Approximation was secured by passing sutures on three surfaces. (*a*) A series of interrupted catgut sutures on the rectal side, penetrating a short distance into the tissue, completely closing the rent in the bowel, shutting off its tract, and thus reducing the tear to a simpler form of vaginal injury. (*b*) Interrupted sutures of silk or silk-worm gut were passed on the vaginal surface, one set extending down to meet the rectal sutures, and between these a set of half-deep sutures. (*c*) A series of sutures on the perineal surface, also deep and half-deep, completely closing the wound area. This old operation is the most satisfactory method ever devised, and yields perfect results.

Second. In another class of cases, comparatively infrequent, where a part of the perineal body was lost without involving the rectum, the denudation was made in a similar manner, and the approximation was the same as in the last operation after the rectal side of the wound had been closed.

Third. The last class of perineal injuries, relaxations of the vaginal outlet, is the most important of all. In these cases the amount of tear, which may be seen after labor, may be very slight, and the scar at a later date may appear very insignificant, but the lax open vaginal outlet, with the prolapsing anterior and posterior vaginal walls, cystocele and rectocele, show at once the entire absence of support to parts immediately above. It must also be constantly borne in mind that the full extent of relaxation is often not apparent at first and can only be discovered by directing the patient to bear down, when the vaginal outlet will dilate and the vaginal wall immediately above will roll down and become everted. Still other cases may even yet conceal the nature of the injury by a constant involuntary effort of the intact fibres of the levator ani muscle, endeavoring to make up the insufficiency of the outlet,—a condition analogous to the spasm of the rectus muscle of the eye in its effort to conceal an insufficiency. Such a condition will be suspected by

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bearing in mind the characteristic pelvic symptoms of pressure, and bearing down, and a sense of weight, dragging and tiredness about the hips, with fatigue upon slight exertion, particularly walking. These patients usually feel most comfortable when in bed. The examination will often reveal a moderate degree of relaxation, but when the patient is thoroughly anaesthetized the levator is relaxed and the full extent of the injury at once appears.

In my earlier work, I was several times astounded upon seating myself to operate upon a lacerated cervix, to discover that I had to deal with an extensively relaxed outlet as well. I know that this is also the experience of other operators, as I have many times had occasion to operate upon the perineum after the cervical operation had been performed by another surgeon. There is no class of cases more important, where the relief afforded is more marked, nor do I know of any cases so universally neglected by gynecologists.

In all cases of relaxation, I perform an operation based upon the principles laid down by Dr. Emmet,—a denudation in front of the rectocele extending up to the caruncles of the hymen on either side and carried up in the form of a triangle into each vaginal sulcus. This procedure forms a natural resection of the over-distended outlet. It yields perfect results, as the condition after a well performed operation closely approximates the virginal outlet.

Silk, silk-worm gut, and catgut sutures have been used throughout. The silk and silk-worm gut on the outside are removed in from seven to ten days; the inside stitches, however, are not touched until from fourteen to twenty-one days after the operation. Sutures in the cervix remain three weeks or longer when the perineum has been operated on and are only then removed with the utmost care to avoid distention of the vaginal outlet.

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Ward No.	No. Days in Hosp.	Name and Condition.	Age.	Color.	Diagnosis.
346	23	J. C. L. M.	35	W.	Laceration of the perineum with relaxation.
259	69	H. H. M.	22	B.	Vesico-vaginal fistula.
355	24	M. E. M.	26	B.	Laceration of the perineum; relaxed outlet.
373	Still in Hosp.	C. E. R. S.	25	W.	Ureteritis; stricture of the ureter; hydroureter.
389	6	M. B. M.	47	W.	Dysmenorrhœa and neurosis of menopause.
396	26	E. C. Y. M.	53	W.	Pelvic peritonitis; uterus imbedded in adhesions on pelvic floor; external hysterorrhaphy a year previously failed to hold it up.
401	13	A. J. M.	27	B.	Lipoma of right labium majus.
417	27	R. D. P. M.	35	W.	Old laceration of the perineum extending 5 cm. up septum.
426	39	V. R. M.	32	W.	Lacerated cervix.
455	11	E. P. M.	27	B.	Endometritis.
466	9	M. G. M.	27	W.	Myxo-sarcoma of the left crus clitoridis.
475	7	A. E. K. M.	42	W.	Cervical polypi.

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Operation.	Suture Material.	Result.	Remarks.
Emmet-Martin.	Silk.	Cured.	
Operation.	Silk.	Perfect recovery.	Entered hospital September 6, '89; remained a month before opera'n.
Emmet-Martin; uterus dilated and curetted.	Silk.	Well.	Complete union.
Catheterization of ureters.		Still under treatment.	*Vide* Art. xiv.
Dilatation and curetting.		Improved.	
Vaginal hysterectomy.		Death.	*Vide* Art. xv.
Removal.	Silk.	Well.	Lipoma, $2 \times 1\frac{1}{2} \times 1$ cm.; primary union.
Bilateral denudation and sutures on vaginal, perineal and rectal surfaces.	Silk in vag. and on perin.; catgut in rectum.	Well; complete union.	Now controls bowel, and has a normal vaginal outlet.
Dilatation and curetting, wedge-shaped excision and approximation of the lips.	Silk.	Union.	Much improved.
Dilatation and curetting.		Improved.	
Incision and enucleation.	Silk.	Well.	Tumor size of pigeon's egg; free hemorrhage controlled by pressure; cavity packed; healing by granulation. *Vide* Art. xiii.
Removal; uterus curetted and packed with iodoform gauze.		Well.	

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Ward No.	No. Days in Hosp.	Name and Condition.	Age.	Color.	Diagnosis.
553	29	K. P. M.	26	W.	Pelvic neuralgia.
547	9	G. P. S.	39	W.	Pelvic peritonitis.
614	36	S. M. M.	34	W.	Laceration of the cervix; relaxed vaginal outlet.
641	57	E. C. S.	45	W.	Adeno-carcinoma corporis uteri.
693	8	C. C. W.	37	B.	Carcinoma of the cervix uteri and vaginal vault.
712	10	E. S. S.	16	W.	Vulvo-vaginal abscess.
714	24	S. B. M.	29	B.	Stenosis of the os uteri and endometritis.
716	13	M. W. S.	31	B.	Elephantiasis clitoridis.
729	40	M. L. M.	38	B.	Laceration of the cervix and perineum.
735	31	A. B. S.	18	W.	Endometritis.
746	20	M. McA. W.	60	W.	Stricture of the urethra.
750	72	F. C. M.	22	W.	Vesico-vaginal fistula.

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Operation.	Suture Material.	Result.	Remarks.
		Improved.	General systemic and tonic treatment.
Topical, counter-irritant and depletive.		Improved.	Had had an oöphorectomy; tender stump and thickening.
Curetting; modified Emmet operation.	Silkworm gut for cervix; perineal sutures silk.	Well.	Both operations at one sitting.
Vaginal hysterectomy.	Silk.	Well.	
Curetted and thoroughly packed with iodoform gauze.	*Vide* Art. xi.	Improved.	Infiltration of broad ligaments; condition much improved on discharge; hemorrhage checked and absence of fetor.
Incised and packed with iodoform gauze.		Well.	Tumor large as pigeon's egg, contents puriform. Inoculation, agar-agar negative.
Dilatation and curetting.		Well.	
Amputation.	Silk.	Well; perfect union.	No suppuration.
Modified Emmet operation.	Silk.	Well.	First operation on cervix, followed a week later by perineal repair.
Dilated and curetted.		Well.	
Dilatation under ether and digital exploration of bladder.	Silk.	Marked improvement in general condition.	Orifice slightly lacerated by fingers; closed by silk; the urethra dilated by bougies, patient directed to continue same daily.
Freshening of edges, 3 operations.	Silk.	Reduction of fistula to diam. 3 mm.	Whole base of bladder involved from cervix uteri into urethra; had undergone six operations previously.

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Ward No.	No. Days in Hosp.	Name and Condition.	Age.	Color.	Diagnosis.
783	39	E. S. S.	16	W.	Endometritis.
794	73	H. H. W.	58	W.	Epithelioma of the whole right labium majus.
811	17	E. C. M.	27	W.	Cystitis urethrocele; incontinence of urine.
814	29	R. W. M.	30	B.	Abscess; inguinal canal and round ligament.
817	30	W. C. W.	42	B.	Laceration of the cervix and perineum, and lipoma of thigh.
827	39	L. G. M.	26	W.	Endometritis.
860	22	J. W. S.	33	B.	Endometritis.
798	43	H. T. M.	28	W.	Laceration of the perineum.
851	47	S. T. M.	36	W.	Complete laceration of the perineum, and dermoid cyst of ovary.
856	38	M. M. W.	31	W.	Relaxed outlet and retroflexed uterus.
917	3	F. R. M.	35	W.	Urethral caruncle.
772	32	L. R. M.	28	W.	Relaxed outlet; retroversion of uterus.

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Operation.	Suture Material.	Result.	Remarks.
Dilatation and curetting.		Well.	Applications and packing continued in dispensary.
Excision of mass.	Silk.	Slight granulating area in the middle.	Wound healed by first intention for greater part; gaped at lower angle owing to tension.
Urethra resected.	Silk.	Union perfect.	Patient left hospital much improved; incontinence not so troublesome; no difficulty while in bed.
Enucleation; and resection of the round ligament.	Buried catgut, and sup. silk sutures.	Well.	Wound gaped at lower angles; healed by granulation. Cavity packed with iodoform gauze.
Modified Emmet; excision of tumor.		Well.	Recovery uninterrupted.
Dilatation and curetting.		Well.	
Dilatation and curetting.	Silk.	Well.	
Posterior median denudation.	Silk.	Well.	Patient very anæmic; operation followed later by oöphorectomy.
Abdominal section and oöphorectomy; Emmet perineal operation.	Silk.	Well.	Double operation, with perfect result.
Modified Emmet.	Silk.	Well.	
Transfixion and excision.	Silk.	Well.	Large pediculated tumor. Had suffered agony during micturition for some years.
Modified Emmet; external hysterorrhaphy.	Silk.	Perfect result.	External hysterorrhaphy, first operation a failure. After second, uterus remained anteflexed several weeks. Examined later in dispensary, uterus back.

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Ward No.	No. Days in Hosp.	Name and Condition.	Age.	Color.	Diagnosis.
775	23	M. L. M.	32	W.	Relaxed outlet.
825	39	T. W.	29	W.	Laceration of the cervix.
791	14	A. M. M.	20	W.	Ovaritis and endometritis.
758	88	G. S.	28	W.	Neurasthenia.
553	35	A. S.	33	W.	Stenosis of os uteri.
546	12	E. M.	28	W.	Chronic ovaritis.
501	97	K. S.	17	W.	Adherent tube and ovary; neurasthenia.
502	14	C. S.	21	W.	Menstrual epilepsy.
539	70	T. W.	61	W.	Carcinoma of cervix uteri.
437	39	S. K. M.	41	W.	Retroflexion of uterus.
374	96	N. M.	42	W.	Laceration of the perineum.

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Operation.	Suture Material.	Result.	Remarks.
Modified Emmet.	Silk.	Well.	
Modified Emmet.	Silk.	Well.	Incipient pulmonary tuberculosis; tubercle bacillus found in sputum; general condition markedly improved on discharge.
Dilatation of cervix and curetting.			
Systemic.		Improved.	
Dilatation of cervix uteri.		Improved.	Cervix torn during gradual dilatation with bougies, opening of cellular tissue in left broad ligament; packed and no ill result followed.
Dilatation of cervix uteri.			
Ovarian adhesions separated by finger per rectum, assisted bimanually.		Improved.	
Palliative.		Improved.	
Vaginal hysterectomy.	Silk.	Recovery.	
Palliative.		Improved.	
Modified Emmet.	Silk.	Well.	Hemorrhage from vaginal wound persisted for some days after operation; partially failed to unite, edges freshened and brought together by sutures under cocaine, followed by perfect union.

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IX.

REPORT OF THE URINARY EXAMINATION OF NINETY-ONE GYNECOLOGICAL CASES.

By HOWARD A. KELLY, M. D., and ALBERT A. GHRISKEY, M. D.

As a part of the thorough investigation of every case admitted to the Hospital, before proceeding to operation, a careful urinary analysis is made. Urine voided naturally is examined first and this examination is immediately followed by a control analysis of urine which is drawn by the catheter. The object of the two-fold analysis thus made is to determine the degree of accuracy, or the necessary fallacies of the analysis of voided urine in the female. It is well known that the contamination of vaginal epithelium, and leucorrhoeal discharges, not to mention the menstrual flow, may prove serious sources of error, either by indicating the presence of albumen in the urine which has merely been added during the act of micturition, or by misleading as to its quantity, and in this way rendering all observations whether qualitative or quantitative more or less untrustworthy. Of this important point I do not wish to speak until a larger number of observations have been accumulated.

A correct knowledge of the condition of the kidneys is clearly of the utmost importance to the gynecologist, inasmuch as the recovery of the patient from any major operation largely depends upon the rapidity with which her emunctories are able to discharge effete products. That renal lesions, sometimes so advanced as to have precluded any possibility of recovery, are found with a surprising frequency in the autopsies of women dying after gynecological operations, is a fact attested by every gynecologist of experience. Two cases in my own experience of unsuspected pyelo-nephritis occurring within the past eight months, also bear witness to this important observation. One of these was consecutive to a hydroureter developed by the pressure of a large fibroid tumor, and died without

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operation (*v.* Art. III.) The other was a pyelo-nephritis complicating a large tubo-ovarian abscess of the left side. During the same time a patient has died in the Kensington Hospital for Women in Philadelphia, after an operation for the removal of densely adherent ovaries and tubes. The autopsy revealed the fact that the kidneys were shrunken, in the last stages of nephritis.

When we recall the many important changes associated with Bright's disease, affecting principally the circulatory apparatus, we will more clearly realize the importance of making the most thorough and painstaking examination of the renal function, lying within our power. Much as it is to be regretted that a urinary examination does not always correctly, or indeed in some cases at all, indicate the condition of the kidney, still under no circumstances is it justifiable to undertake any major operation without such careful analysis.

Just here I am inclined to think, will be found an important gauge of that mysterious factor "vitality," so much talked about. Here lurks a veritable danger which will often unexpectedly carry off patients apparently in blooming health. The "vitality" of a patient certainly is not a single physiological entity, but is clearly the resistance offered by the system at large to the destructive tendencies of disease or injury, and stands in direct ratio to the healthy condition of each organ in the body which is directly concerned in the process of tissue building and the discharge of tissue waste. Let there be but a slight damage to one of the kidneys and effete products will tend to accumulate in the body.

A knowledge of the condition of the kidneys is clearly of practical value for the following reasons:

First, that we may refuse to operate upon cases presenting advanced renal lesions.

Second, that we may in some cases delay the operation to try the effects of preparatory treatment—of rest, diet and medicine, and quickening the action of the other emunctories.

Third, that we may adopt unusual precautions in the course of the operation complicated by kidney disease, preferring chloroform to ether anæsthesia, avoiding undue prolongation of the operation, and

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particularly in using the drainage tube to lessen the tax on the absorptive powers of the peritoneum.

Fourth, that we may watch such cases carefully throughout the convalescence, avoiding with especial care the use of opiates, and endeavoring to make up for the crippled action of the kidneys by calling on the skin and bowels to do supplementary work, and, above all, avoiding the sudden checking of a diarrhœa.

Fifth, that a careful urinary examination will sometimes lead us to operate at an earlier date, for we are able thus to discover incipient disease of the kidneys, depending for its cause upon pelvic obstructive disease pressing upon the ureters, thus forecasting the future development of more serious renal disturbances.

Guided by these principles, I have, in a number of instances, hastened an operation for fibroids or pus sacs, which were pressing upon one or both ureters—cases in which the operation would have been postponed had not this serious danger signal been recognized.

It has happened in several instances that the albumen has entirely disappeared from the urine within a few weeks after the operation, while the patients were still in the hospital; this was well shown in the case of a large ovarian cyst (No. 34 in the list of laparotomies). The urine contained a marked albuminous ring which disappeared entirely after the operation.

The following were the methods employed and the results obtained:

Up to the end of December, 1889, Heller's test for the detection of albumen was used in most cases in analyzing the urine of gynecological patients.

About this time, as the value of the trichloracetic acid, as a more delicate reagent than nitric acid, was being demonstrated in the Clinical Laboratory by Dr. D. M. Reese,[1] of the medical staff, it was used conjointly with the older method until February, 1890. In a number of cases, traces of albumen were shown by trichloracetic acid, where with nitric acid it was either doubtful or negative, and in these a careful microscopic examination revealed also the presence of casts or leucocytes. The comparison between the two acids was made directly after the urine was brought into contact with the reagents. In several instances the presence of albumen was

[1] See *Johns Hopkins Hospital Bulletin*, No. 3.

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not shown by Heller's method until some time after the application of heat. The promptness of the reaction with trichloracetic acid, as well as its delicacy, commends its use.

Since the beginning of 1890, an examination of both voided and catheterized urine has been made in all cases for the purpose of detecting any albumen due to vaginal discharge mixing with the urine in the act of micturition. These results will not be published at present.

The urine is tested for albumen and casts several times after the operation in each case.

Ether is usually used as an anæsthetic; chloroform being employed when renal disease is present, and in cases of pulmonary disease, especially bronchitis, as well as where there is a tendency to choke much or to constant coughing. In no case has any untoward effect followed the use of chloroform. The existence of valvular cardiac disease has not been considered a contra-indication to its use.

Ninety-one gynecological patients were examined with reference to the presence of albumen and casts in the urine previous to operation.

Specimens of catheterized urine in fifty of these, which were cases of abdominal section, showed traces of albumen in fifteen instances; in eight of the fifteen, hyaline and granular casts were found.

In two cases of vaginal hysterectomy, a trace of albumen with hyaline casts was present in one, while the second was negative.

In forty-one gynecological cases remaining, other than abdominal sections, albumen was present in twelve, usually a faint trace, with casts in seven of the twelve. In the six cases which died, two without operation, albumen and casts were found in four, one was negative, and one not examined.

In three instances in which traces of albumen with hyaline and granular casts were found, two of pyosalpinx and one of ovarian cystoma, albumen and casts could not be detected just before patients were discharged from the hospital.

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X.

LIGATURE OF THE TRUNKS OF THE UTERINE AND OVARIAN ARTERIES AS A MEANS OF CHECKING HEMORRHAGE FROM THE UTERUS AND BROAD LIGAMENTS IN ABDOMINAL OPERATIONS.

By HOWARD A. KELLY, M. D.

It occasionally happens in the course of an operation involving the broad ligaments, or the cornua, or lateral walls of the uterus, that the tissues are extensively torn, or so decomposed as to break down under the fingers, leaving a broad, ragged, freely-bleeding surface which it is very difficult to control.

Tying off bleeding vessels on the lateral uterine wall is particularly difficult on account of the flat surface, and the resistance of the muscular tissue which does not allow the vessel to be drawn out far enough to make a secure tie, and it is often equally difficult to find the oozing points in a rent in the broad ligament. To obviate this difficulty I have repeatedly tested, and found the following expedient of the utmost value, both rapidly checking the flow of blood leaking from a multitude of small vessels, and insuring immunity from hemorrhage after the abdominal incision is closed.

Where the lateral surface of the uterus is widely laid bare *a ligature may be applied to the main trunk of the artery* near the base of the broad ligament, securing both the common uterine artery and its branches, thus completely cutting off the flow of blood to the area above.

This may be done in the following manner: the uterus must first be drawn towards the opposite side, thus giving a good exposure of the wound area; when a stout curved needle, armed with strong silk or catgut a foot long, is carried from ½ to 1 cm. beneath the lower limit of the tear, just entering the substance of the uterus and pene-

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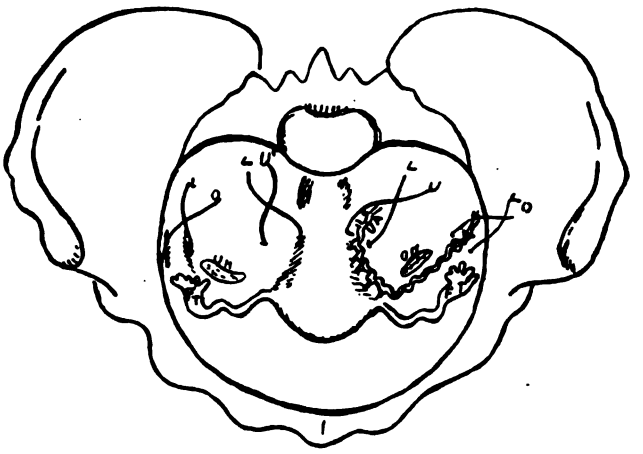
trating both layers of the broad ligament; it is carried back through the broad ligament about 1 cm. to the outside of this; the ligature thus lies around the uterine artery, and is tied in the intact portion of the injured broad ligament and the hemorrhage ceases.

If active hemorrhage continues at the fundal end, it may be necessary to cut off anastomosis, by *ligating both uterine arteries* in a similar manner.

FIG. 1.



Showing points at which the main trunks of the uterine and ovarian arteries may be ligated.

F. T. Fallopian tube.
O. H. Ovarian hilum, ovary removed.
U. A. Main trunk of uterine artery in pelvic floor.
O. A. Trunk of ovarian artery in infundibulo-pelvic ligament.
L. U. Ligature placed around trunk of uterine artery.
L. U'. Ligature shown placed in a position in which it is sure to grasp uterine artery.
L. O. Ligature placed around ovarian artery, beyond the extremity of the tube, and in the infundibulo-pelvic ligament.

Where there is no injury outside of that to the uterus, and the tie is made in the intact broad ligament, the recto-uterine and vesical pouches must be exposed to view; when upon determining the lowest part of the vesico-uterine pouch with the eye, while grasping the uterus and holding it well up, the ligature needle is carried through the broad ligament from before backward as close as possible to the uterus. It is then passed back through the broad ligament one centimeter outside of the first puncture, and tied. In this way both uterine arteries are readily controlled (*v.* Fig. 1, L. U'., L. U.).

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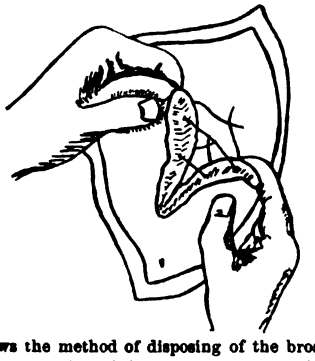
The ovarian arteries can be easily ligated by carrying the needle through the well-defined infundibulo-pelvic ligament as it courses

FIG 2.

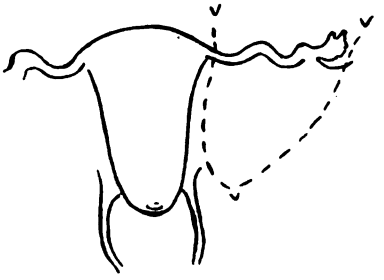
V. Vagina.
C. Cervix uteri.
B. L. Broad ligament opened on side of uterus.
b. l. Base of broad ligament extending from cervix uteri out to pelvic wall.
l. Catgut ligature embracing uterine artery and checking hemorrhage from B. L. above.
U. A. Uterine artery, ligated in the intact broad ligament by ligature l.

FIG. 4.



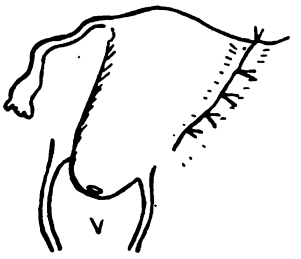
Shows the method of disposing of the broad raw surfaces left by excising the wedge in Fig. 3. The result is shown in Fig. 5.

FIG. 3.



Shows wedge sacrificed in left broad ligament, drawn together in Fig. 4, completely closed in Fig. 5.

FIG. 5.



Result of suturing the base of the broad ligament on the uterine wall, to the base on the pelvic floor and pelvic wall.

outwards and backwards, just beyond the fimbriated extremity of the tube, and tying over the top of the ligament (*v.* Fig. 1, L. O.).

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I have resorted to this means of checking an obstinate hemorrhage at least seven times, and it has not failed in a single instance. After practising it once or twice the operator will feel that outside of cases of large fibroids choking the pelvis, where it is impossible to reach these vessels, he is always master of the situation as far as hemorrhage is concerned.

I have never yet seen any evil effect from thus cutting off the blood supply of the uterus on both sides.

The diagramatic sketches will explain themselves.

Where through extensive attachments of the tumor to the broad ligament, or intraligamentous growth, or through necrosis of the mass including much of the broad ligament below it, the ligament is extensively or wholly sacrificed, such an area being removed as is shown in Fig. 3, v, v, v, after controlling the hemorrhage in the manner described, the large wounded surface of the whole broad ligament still remains, and if simply dropped back into the pelvis in this way will certainly contract adhesions with the surrounding intestines, with danger of an ileus at a later date.

This difficulty may be avoided by the simple device of attaching the base of the broad ligament on the lateral wall of the uterus to that part extending from the cervical portion of the uterus out to the pelvic wall (*v.* Figs. 4 and 5).

The introduction of a single suture as shown in Fig. 4, where the uterus is grasped in the left hand and the infundibulo-pelvic ligament in the right hand, is sometimes sufficient to secure the approximation.

The result of thus disposing of the raw surface by doubling it in on itself, is to produce such a marked latero-flexion of the uterus, as is shown in Fig. 5.

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XI.

CARCINOMA OF THE CERVIX UTERI IN THE NEGRESS.

By J. WHITRIDGE WILLIAMS, M. D.

The comparative immunity of the colored race from cancerous diseases, together with the marked predisposition to the formation of fibrous growths, is well recognized by all experienced clinicians, and is a fact needing no further demonstration. This immunity not only extends to the uterine tissues, but is even more markedly emphasized there than in any other part of the body.

No case of cancer of the uterus has as yet been recorded in the negress, and even cases occurring in mulattoes are accounted rarities, although the victim may be half or almost entirely white in color, and therefore, strictly speaking, not entitled to rank as a negress.

Emmet, in speaking of cancer, dwells upon the fact of its rarity in the negro, stating that in all his experience he has seen but one case of carcinoma uteri, and that was in a mulatto.

Schroeder quotes Whitall of New York as stating that of 2,000 colored women treated by him, in only two was there a malignant disease of the uterus, and in the absence of a more definite statement these were probably mulattoes.

During the past four years I have seen three cases of carcinoma uteri in the mulatto; and, until the present case came under observation, I believed, in common with others, that the pure black enjoyed an absolute immunity from the affection.

That this is the general impression throughout the profession will be evident upon looking through the literature of the subject. I have been unable to find a report of a single well authenticated case.

It is, however, not improbable that the report of this case will call forth others, as in the instance of ovarian cystomata, a few years since.

The following case occurred in the service of Dr. H. A. Kelly, in in the gynecological ward of the Johns Hopkins Hospital.

On Jan. 9th, 1890, C. C., a dark, swarthy-skinned, full-blooded negress, was sent by Dr. Steele, of Cambridge, to the Hospital.

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She was 38 years old, and had married sixteen years ago. In this period of time she had borne eleven children, the youngest being 9 months old. She had never miscarried. All her labors were easy and normal, and she had nursed all the children.

Except when pregnant or nursing, the menses appeared regularly, causing her no anxiety until last July, when she began to "menstruate daily." This occurred three months after the birth of the last child. The loss of blood, at first slight, became freer, and was at times very profuse. The discharge was, as a rule, dark and lumpy in character, but at times thin and pale.

At the time of admission she complained of the constant flow, and severe pain in abdomen and back.

The following day she was examined under ether. The vaginal outlet was found relaxed, and the vagina smooth. A large excavation existed at the vaginal vault, where there was a deep pit in the position of the cervix, 5 cm. in diameter, extending on the left side into the broad ligament and 3 cm. up into the uterus. The excavation was studded above and on its right margin with slightly elevated granulations. The vaginal margin, limiting the cavity, was sharply defined and appeared healthy. The uterus was immovable, with its corpus thickened and anteflexed.

Upon rectal examination, a broad mass of infiltration was found extending into both broad ligaments. On the right side a large mass of infiltration was firmly attached to the pelvic wall. On the left side the whole broad ligament was densely infiltrated from the uterus to the pelvic wall.

The history and examination at once settled the diagnosis as carcinoma of the cervix uteri and of the broad ligaments, the latter being involved by the extension of the disease in the usual way. Vaginal hysterectomy was totally out of the question, and the utmost that could be done was palliative treatment by curetting. About half an ounce of scrapings were removed with Simon's sharp spoon, the outer tissue breaking down like cheese; much of the rotten tissue was removed also by the fingernail, until a hard base was reached, leaving a conical excavation, extending up to the internal os. Beyond this the uterine cavity measured 4½ cm. in depth.

A piece of the tissue on the vaginal margin was excised for microscopic examination. The cavity of the uterus and the vagina were packed with iodoform gauze.

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After this operation the patient made a perfect recovery, and left the Hospital on the seventh day to return to the country.

On careful microscopic examination, both the scrapings and excised bit of tissue confirmed the diagnosis of carcinoma. The entire section was densely infiltrated with epithelioid cells, accompanied by a large amount of round cell infiltration. The epithelioid cell infiltration was so abundant that it had almost entirely replaced the muscular tissue, which was represented here and there by a few scattered fibres.

A second case has been under treatment in the out-patient department of the Hospital.

March 12, 1890, C. J., colored, aged 48 years.

Married thirty years; has had four children, the oldest 22 and the youngest 16 years of age; one miscarriage 30 years ago. The labors were easy and uncomplicated. The menopause occurred four years ago, and up to one year ago she lost no blood whatever; but since that time she has had a more or less constant loss of blood, accompanied by a fetid discharge in which shreds of tissue were seen. The bowels have been irregular and she complains of great pain in the back and lower abdomen, and for the past few weeks she has had frequent and painful micturition.

Examination.—On passing the finger up the vagina, at a distance of 4 cm. a deep cavity is reached, $2\frac{1}{2}$ x 3 cm. in diameter, with rough granulating walls and indurated edges, occupying the position of the vaginal portion of the cervix and extending into the fornices. The uterus is retroposed, enlarged, sensitive and firmly fixed by dense infiltration extending into the parametria and involving the posterior wall of the bladder.

As the case had progressed too far for operation, palliative applications of iodoform and tannic acid were made. Unfortunately there is no further history of the case as the woman returned but once to the dispensary.

On being questioned as to her color, she stated that she was a brown negress and that her father was of the same color as she, while her mother was pure black. Her father's color was too dark to admit of either of his parents being white, and thus the nearest possible contamination with white blood must have taken place in the time of the patient's great grandparents, and most probably at a more remote period.

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XII.

ELEPHANTIASIS OF THE CLITORIS.

By HOWARD A. KELLY, M. D.

M. W., African, age 31, single, admitted to Hospital Jan. 14, 1890. Diagnosis—Elephantiasis of the clitoris. Excision. Discharged Jan. 27th, cured.

The patient was a poorly nourished negress. She menstruated first at 13 years of age, moderately and at regular intervals. Menstruation now is irregular, lasting from three or four days to several weeks. The flow is very profuse and accompanied by little pain. She has a constant free leuchorrheal discharge, and complains of pain in the small of the back and the lower abdomen, with cramps in the legs. She has also a swelling in the privates which causes constant pain. She is very constipated, and troubled with frequent micturition, worse at night.

Examination.—The cleft of the vulva is distended and filled out with a large tumor, which is pendent from the anterior commissure. On lifting this up, the vaginal outlet is found relaxed. The cervix uteri is stellately torn, and the uterus lies retroposed in the sacral hollow. The tumor hanging between the thighs and covering the genitals, occupies the position of the clitoris, and is closely connected with the labia minora. Its lower extremity is rounded and free, slightly notched underneath, looking like a large glans penis with a retracted prepuce, having a well defined sulcus back of the corona. The upper end of the tumor is attached by a broad base over the symphysis pubis. The whole length of the mass is 10 cm.; its greatest breadth 5 cm., and its thickness, antero-posterior, 4.5 cm. Both of the nymphæ above are much thickened and form part of the base of the tumor, covering its dorsum and forming the thickened preputial fold encircling the glans. On raising the mass, the urethral orifice is found intact. The whole vaginal orifice is thickened and

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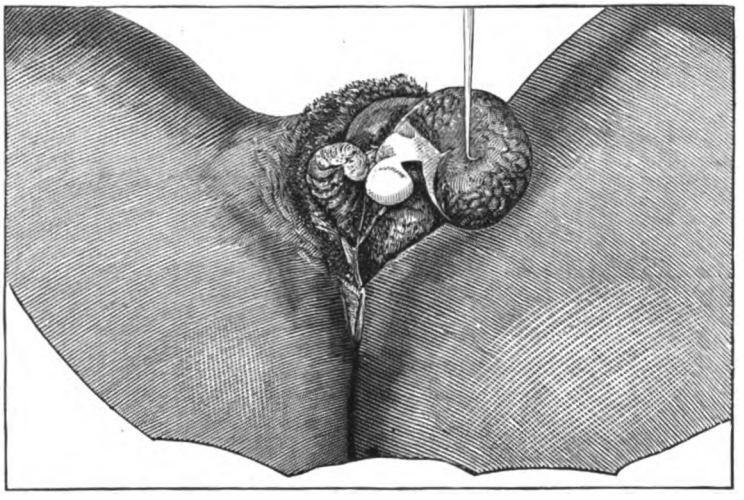
corrugated and several superficial rounded areas of ulceration are visible, looking like mucous patches from ½ to 1 cm. in breadth. A fetid leucorrhœal discharge issues from the vagina. On the dorsal surface of the tumor is an irregular patch of white, contrasting markedly with the deep pigmentation of the rest of the mass. At the junction of the right nympha with the tumor, arise two smaller pediculated tumors, the size of a pea and a hazel-nut respectively. Scars in the left groin and in the supra-clavicular region, are evidences of old syphilitic disease. The case, therefore, is one of syphilis, with elephantiasis involving the upper portion of both labia minora and the body of the clitoris.

ELEPHANTIASIS OF THE CLITORIS.

The enlarged clitoris is seen drawn to the left side by the tenaculum.



Operation, Jan. 15, 1890, in the amphitheatre of the Johns Hopkins Hospital. Patient anæsthetized and after thorough cleansing, and as thorough disinfection of the parts as was possible, I commenced the removal with the knife. Grasping the mass in the left hand and drawing it well out from the body, a distinct pedicle was formed which was transfixed at its base with a stout curved needle, armed with a strong silk suture. After three sutures were introduced, one below the other, the corresponding part of the tumor was cut away from above downwards, and slightly cupped in the centre, and then the sutures being rapidly tied were made to serve the double purpose of approx-

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imating the opposite edges of the incision, and controlling hemorrhage from the brawny tissue. By successive steps, first introducing a few sutures, then cutting away the overlying mass and drawing the sutures up, and then passing more sutures, cutting the pedicle again, and so on, the whole mass was quickly removed with but trifling hemorrhage. Below, the base of the tumor diverged where the nymphæ separated from the clitoris, thus making an inverted Y-shaped wound, 6 cm. long.

The wound was dressed with iodoform and boric acid powder, and healed without suppuration. 1–16–90.—Wound dry. Dressing changed. Patient comfortable. No pain. 1–18–90.—Doing well. Wound perfectly dry. 1–22–90.—Stitches removed. 1–27–90.—Discharged well.

Pathological Report.

An irregular long mass, firm, more or less constricted in the middle. Uniformly deeply pigmented with the exception of one area 2 x 1 cm., which is perfectly free from pigment. Its upper portion contains numerous irregular teat-like projections; on its under surface is a more or less varicose arrangement, and at one point on under surface, a superficial loss of substance with firm smooth edges. The whole mass bears some general resemblance to the shape of the clitoris, measures 7 x 4½ x 3½ cm. On section, not very vascular, soft œdematous tissue, fluid easily squeezed from cut surface.

Diagnosis.—Elephantiasis of clitoris.

Sections were made through the tumor at several points which show the histological appearances to differ somewhat in different places in the growth. On the under surface of the tumor, sections were made embracing the ulcerated surface immediately beneath the tumor and extending through it, so as to take in the vascular supply at the place of attachment.

The ulcerated surface shows an intense small-cell infiltration with numerous blood vessels which extend three to five mm. below the surface; the cells here are generally round with little or no protoplasm surrounding them, and densely packed together, passing gradually into a looser connective tissue, with marked cellular infiltration around the blood vessels. Immediately beneath the epidermis there is a dense connective tissue composed of broad bands of fibres,

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becoming looser towards the middle of the section, where there is a good deal of cellular infiltration, chiefly along the vessels—all of the appendages of the skin, sweat glands and a few sebaceous glands are contained in this. The sweat glands are surrounded by a dense, firm connective tissue with very few cells, some are dilated, the epithelium markedly thickened and showing some atypical growth. The vessels going to the tumor, included in this section, have very large and thick muscular coats, and in some the intima is also thickened. The nerves are preserved, some show no alteration, in others there appears to be an increase in the connective tissue. The epidermis is unaltered.

Sections made from the surface of the tumor involving one of the unpigmented areas, show an entire absence of pigment over this spot. At one point there is a focus of cellular infiltration immediately beneath the epidermis with numerous leucocytes in the epidermal layers; between the cells the connective tissue in the tumor throughout shows about the same character, it being arranged in large refractive bands. The connective tissue spaces are increased in size, and the cells generally larger than normal—in looser parts of the tissue this increase in size of cells is particularly marked. Cells are found with two or more nuclei, and here and there, scattered in places through the section, are large giant cells, lying usually in the interfibrillar spaces, irregular in shape, frequently containing large vacuoles; the nuclei in them are large and oval, rather pale, sometimes irregularly distributed throughout the cell, and in others arranged around the walls. The small cell infiltration is not so marked as in the preceding specimen. There appears to be some increase of pigment in certain places. The pigment is found in some of the connective tissue cells immediately beneath the epidermis, but especially in long lines which extend between the epithelial cells of the lowest layer for a considerable distance into the epithelium, these lines presenting here and there slight varicosities, giving the impression of channels filled with the dark-brown pigment. No cells are seen in connection with these pigment lines. Scattered through the tissue, and seen in all the sections, are large round masses with refractive globules which stain slightly with hæmatoxyline and with all the analine stains—darker with the latter—in none of these are any nuclei found. Although the lymph spaces in the specimens are generally dilated, no distinct lymphatic vessels are seen.

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XIII.

MYXO-SARCOMA OF THE CLITORIS.

By HUNTER ROBB, M. D.

The report of this case is of interest on account of the infrequency of tumors of the clitoris.

Mrs. G., age 26, Pole, married, applied to the Gynecological Department for treatment, and was admitted to the Dispensary, September 17th, 1889. She complained of a constant pain about the external genitals, increased by coitus.

Upon examination a tumor was discovered on the left side overlying the upper part of the descending ramus of the pubis, $4\frac{3}{4}$ cm. long and $2\frac{1}{2}$ cm. in width, pointed at either end, hard, movable, and slightly lobulated; it occupied the position of the left crus clitoridis, and apparently had its origin in the substance of the clitoris itself.

The patient was at once urged to enter the Hospital for operation, but refused, requesting the removal of the tumor in the Dispensary, which was acceded to as a simple enucleation was anticipated. After injecting the integument immediately over the surface of the tumor with several minims of a 4 per cent. solution of cocaine, an incision $2\frac{1}{2}$ cm. long was made over the most prominent portion, and the tumor, which was contained in a fibrous capsule, was enucleated without much difficulty. There was scarcely any pain felt during the operation, but a free uncontrollable venous oozing followed, necessitating pressure and packing the cavity with absorbent cotton, permeated with tannic acid.

On account of the loss of blood, and the fear of subsequent hemorrhage, the patient was induced to enter the public ward. From the day following the operation, until her discharge, seven days later, the cavity was daily washed out with a watery solution of 1 to 1000

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bichloride of mercury. At the time of leaving the hospital, the wound had contracted down to a small linear wound without any suppuration.

Pathological Examination.

The tumor is an oblong, smooth, slightly lobulated mass, with thin and loose connective tissue adhesions; it is of firm consistency, of grayish-white color and elastic, the cut surface dry.

The greatest length of the tumor is 4½ cm. by 2¼ cm. wide.

Sections, after hardening in Müller's fluid, show that the tumor is composed of two sorts of tissue; of cells lying in groups or in long rows with a smooth homogeneous substance between. In some portions of the tumor instead of cell groups there are single cells with long, irregular processes, communicating with each other, and embedded in the homogeneous material which fills the intervals between the cells. The cells vary in shape, those in the groups and long rows are generally spindle-shaped, with long nuclei; some of the nuclei resemble closely those of non-striated muscular fibres. All of the cell groups and bands communicate with each other, so that the homogeneous material between them appears as small islands. In some portions of the tumor the cell groups are large and make up the greater portion of the tissue, and every gradation can be seen between these places and the single cells embedded in the tissue. In still other places the intercellular substance predominates; there being only scattered nuclei in the homogeneous material with very little cell substance around them.

In sections of the tumor made after freezing, the homogeneous material swells up and becomes perfectly transparent on addition of acetic acid. In the hardened sections it is in places slightly granular and stains faintly with eosin.

The blood vessels are few in number, small, and are always found in the homogeneous material, their walls gradually passing into this substance.

In sections of the tumor stained in picro-carmine, the long bundles of cells, where the nuclei are longest, stain a bright yellow like muscular fibres, and remind one very much of muscle cells; nothing of this can be made out in other portions of the tumor.

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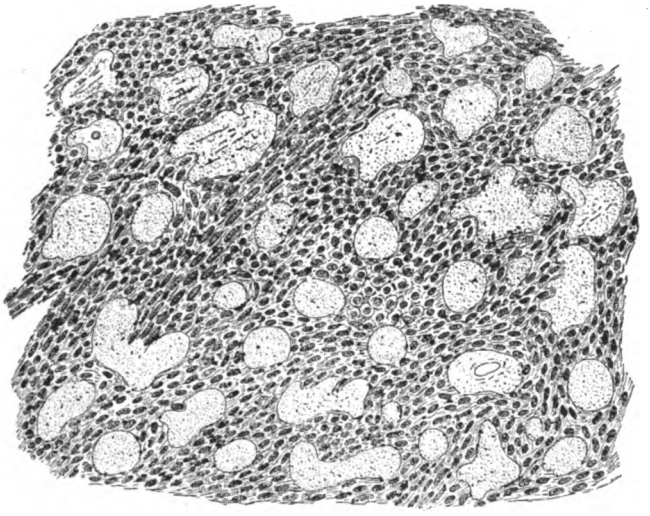
The tumor is a sarcoma, the homogeneous intercellular substance being formed by myxoid degeneration of its tissue, therefore a myxo-sarcoma.

FIG. 1.

Tumor ¾ natural size, showing lobulated condition.

FIG. 2.



Section of tumor after hardening in Müller's fluid, and staining with haematoxyline and eosin, x 80.

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XIV.

KOLPO-URETEROTOMY.—INCISION OF THE URETER THROUGH THE VAGINA, FOR THE TREATMENT OF URETERAL STRICTURE.

By HOWARD A. KELLY, M. D.

Diseases of the female urinary tract have always been treated rather after the fashion of a step-child than a legitimate member of the gynecological household, and beyond a familiarity with the signs of calculus, and of cystitis, and of the surgical treatment of vesico-vaginal fistula, the average gynecologist does not advance. This is unfortunate, inasmuch as this branch of gynecology already has an important past, embracing an extensive literature of some of the best work which has yet been offered to the surgical world.

With Skene's discovery of the existence of the urethral tubules, important lurking points for recurring pus-inoculations, and Emmet's description of the diseases to which the urethra is liable, and the work of Simon and Winckel on the female bladder, and that of Pawlik and Sänger on the catheterization and the palpation of the ureters, it will be seen that we have a complete chain of investigations extending from the meatus urinarius externus all the way up to the kidneys.

It is my purpose here to draw attention to several points of importance in ureteral pathology.

The earliest effort made to catch the secretion from one of the kidneys before it became so mixed in the bladder with the discharge of the other that differentiation was impossible, was by Tuchmann, who cut off the secretion of one kidney while collecting that of the other, by introducing an instrument like a lithotriptor into his own bladder opening the blades, and rotating it to the position of the ureteral orifice, when on closing the blades, the ureter was thus pinched and occluded. This method proved unsatisfactory, and on account of the manifold difficulties and dangers unfit for a wider field of usefulness.

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Silbermann next tried to attain the same end by imitating a pathological process which he had observed. A movable pediculated tumor in the bladder near its neck had by its weight in the course of time, produced hydroureter. Silbermann therefore attempted to imitate this pathological process and to shut off one ureter for a short time, while collecting the urine passing through the other, by introducing into the bladder a flaccid bag on the end of a catheter and filling it with mercury, and then by changing the position of the patient to cause the bag to fall on one or the other ureteral orifice, and thus temporarily occlude it. This attempt also failed, and but little more was accomplished until the appearance of G. Simon's work, demonstrating the practicability of dilating the urethra, after incising the outlet, sufficiently to admit an index finger, which then palpated the ureteral orifices within the bladder, and served as a guide for the introduction of a catheter into the ureters.

It was some years later when Pawlik first demonstrated in Vienna, the perfect feasibility of catheterizing the ureters free-handed through an undilated urethra, without any preparation or guide other than the eye watching the point of the catheter within the bladder, as it plays over the anterior vaginal wall seeking the orifice of the ureter, known approximately by certain vaginal landmarks.

Sänger's work in the palpation of normal and diseased ureters through the vaginal wall, demonstrating the practicability of an examination of this portion of the urinary tract as a routine procedure, added the one important step necessary to give completeness to our means of investigation in this field.

Clear and satisfactory as these demonstrations have been, the work has received but little attention.

I offer the following case as a contribution to the pathology and surgical treatment of diseases of the ureters.

Stricture of the left ureter, diagnosis by catheterization. Treated by opening the ureter through the vaginal vault, and dilatation.

The history of the case previous to coming to me is given largely in the patient's own words.

Miss X., twenty-six years of age, single.

The present disease began when she was fifteen years old, when without apparent cause the bladder became irritable and was unable

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to retain the urine a moment after the desire was felt to expel it. This ceased after a month, to return at each menstrual period. It continued thus menstrual in character for six months, and then began to extend beyond the period. Four months later there was pain in the bladder all the time, confining her to the bed-room, helpless from the distress, which was increased by walking or moving. Menstruation then disappeared for six months, returning after a course of treatment for misplaced uterus. This relieved the constant pain, but the frequent micturition remained. A hard rubber catheter worn in the urethra for ten weeks did no good. After a year's suffering, during which time innumerable prescriptions were tried, the bladder was washed out twice a week with warm water. No improvement followed the washing. Another year passed, during which time a pain was added in the left side, beginning in the loin, extending into the hip, and down the leg.

The endoscope was then used without ascertaining anything. Distention of the bladder with water and afterwards with gas, was also practised, but caused such increase of the pain that it was soon discontinued. Electricity was used for four months without benefit.

At this time the suffering in the side began with a rolling of gas in the bowels, which moved frequently in the night, ceasing only when the bowels were emptied, after three or four loose movements, early in the morning.

For four years passages from the bowels always brought on distress in the bladder, which lasted an hour or longer. The stomach gave out next, even simple food causing much distress, followed by the pain in the side and the looseness of the bowels. The dyspepsia was improved after a year by change of climate, but the bladder remained as bad as ever, being still unable to retain more than a small quantity of urine, and giving great distress when emptied. Three and a-half years ago she was confined to her room with acute pain in the bladder, and a sensation of contraction in the rectum inducing the desire to urinate constantly; the menstrual periods also became very scanty. Treatment for a misplacement of the uterus for eight months did no good. Pessaries could not be kept in place at all, being forced down by the spasms of the rectum and bladder.

She now came into the hands of a very able specialist, who treated her for an ulceration of the bladder, which improved under washes,

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and the irritation in the rectum also subsided, and she was able to get about with some comfort for three months, although still troubled with loose bowels, and the old suffering in the left side and extreme vesical sensitiveness. Continued washing failed, however, to give permanent relief, and at the end of three months the rectal irritation returned, along with a sharp and shooting pain in the bladder, and a sense of "tight constriction in the rectum" and "rigid bound up sensation." Stricture of large intestines was thought to be the cause of this, and medicine was given every other morning to open the bowels freely. After a month this treatment was followed by a puriform discharge, thought to come from an abscess connected with the bowel. During all this time the patient lost both flesh and strength, and suffered more than ever with the spasms in the rectum and bladder. An operation was then planned, but never carried out, to separate adhesions, which it was thought must bind the upper part of the bladder to the intestines.

The bladder was washed out for a year and a-half continuously without any final improvement.

A year later both ovaries and tubes were removed, as up to this time the symptoms had always been so greatly intensified about the menstrual period, that it justified the expectation that much relief would be afforded by the menopause. The ovaries were found enlarged and cicatricial, with a dense white capsule. For two weeks after the operation all spasmodic pain ceased, although she still urinated with pain, about every half hour. After two weeks of comparative rest, however, the old trouble of loose bowels and irritated bladder returned. At the end of three weeks a fever came on lasting for several days, with tenderness over the kidneys, and increased irritation at the bladder. This fever and tenderness disappeared within a few days, but the old rectal and vesical difficulties remained.

She came into my hands in the spring of 1889, through the courtesy of my friend Prof. M. D. Mann, of Buffalo, who believed her sufferings to be due to ureteral disease, and knowing my interest in this work, sent her to Philadelphia.

I found a pale, but fairly well-nourished blonde, confined to her room by constant micturition, and the severe spasms accompanying the act. She suffered such agony at times that she screamed loud

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enough to disturb the whole neighborhood. The urine passed was of a clear, lemon-color, depositing a thick mucus on standing. It contained no albumen or sugar, nor any evidences of cystitis. The hyperæsthesia of the whole genital tract was so great that it was necessary to use an anaesthetic to make the most superficial examination. The vagina and uterus were found to be normal, and the parametrium free from scars or deposits. But a careful palpation of the anterior vaginal wall revealed a cord-like structure lying in the cellular tissue on each side anteriorly sweeping around the lateral pelvic walls, from the neighborhood of the cervix uteri posteriorly, to the neck of the bladder behind the symphysis pubis in front.

The cord of the left side was most distinctly felt; it seemed to be as large as a lead pencil, as it rolled under the palpating finger. The location and direction of these structures left no doubt that they were the right and left ureters, and thus both ureters could be traced for about 10 cm. from the cervix downwards, gently curving and easily displaced in the loose pelvic connective tissue.

The left ureter was hard and nodulated, as if slightly constricted at irregular intervals.

In the left fornix close to the cervix, where the left ureter passed under the broad ligament, a distinct hard, relatively immobile mass was felt, such as I have several times seen mistaken for an inflamed adherent ovary.

When not under an anaesthetic, these ureteral cords were so exquisitely tender that the gentlest palpation caused her to scream with pain, and pressure induced an intense desire to urinate.

I proceeded at once to catheterize the ureters, first emptying the bladder of an ounce of clear urine, and then distending it with six ounces of a weak saline solution, to get rid of the folds in the mucous membrane. She was then brought into the lithotomy position with the hips over the edge of the bed and knees and thighs well flexed; then introducing the index finger of the left hand into the vagina, I ascertained the point at which the cord-like extremity of the left ureter ended in the bladder. With this as a guide, the ureteral catheter[1] was

[1] I use Pawlik's catheter modified, replacing the slit by a series of holes. The sharp-edged slit was very apt to catch and tear the delicate mucous folds of the urethra.

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introduced into the bladder, lightly poised between index finger and thumb, and with its concavity directed outwards it was gently swept downwards, backwards, and outwards, in the search for the ureteral orifice. The characteristic tripping sensation, produced by the end of the catheter jumping over the ureteral prominence, was soon recognized, and by gently playing the tip of the catheter to and fro at this point it engaged and took its own course, no longer guided by the finger, and swept outward and backward along the pelvic wall. On withdrawing the plug in the end of the catheter, a few drops of clear urine escaped, and after a few seconds a few drops more were discharged, and an intermitting flow was thus kept up. Upon attempting to withdraw the catheter there was distinct resistance, and it was found firmly grasped at a contracted point within the ureter. The right ureter was also catheterized without difficulty, but no stricture was found. After this examination she suffered acutely for several days, during which time it was necessary to give ½ gr. doses of morphia hypodermically at frequent intervals to secure quiet. When suffering she assumed a peculiar attitude, sitting up in bed on the rubber cloth, crouching and hugging her knees whenever the spasms came on.

I catheterized her in this way about six times, at intervals of from ten to twelve days, each time under an anæsthetic, each catheterization being followed by a few days of marked exacerbation of the pain, but later by a period of very marked relief. Sometimes the catheterization was effected by Pawlik's method, introducing a speculum and retracting the posterior vaginal wall while the catheter was being introduced. No other treatment was employed beyond these dilatations of the pelvic ends of the ureters.

She returned home in June, 1889, so much improved that she felt almost entirely well, being able to go about and to take long drives. In November she came to me in Baltimore, with some return of the old symptoms.

I found that the ureters were still large and tender. By rectal palpation I was unable to detect any enlargement of the ureter above and beyond the left broad ligament. To dilate the stricture in the left ureter more effectually, as well as to examine the ureter and penetrate higher than is possible with a metal catheter or sound, I had a whalebone ureteral catheter constructed by Mr. Lentz of Philadelphia. Upon introducing this instrument into the left ureter, it was

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again distinctly felt to be grasped under the left broad ligament. This was easily demonstrable to visitors and assistants who were present.

The first dilatation of the stricture by the whalebone bougie was followed by a week of severe pain accompanied by frequent rectal and vesical spasms, making it necessary for her to sit day and night on the bed-pan. Every few minutes, when the spasms came on, she would scream, disturbing all the patients on the same floor. After this there was marked temporary improvement, which did not increase under subsequent dilatations.

Finding after two months of treatment that I was unable to relieve the severe suffering, I opened the base of the bladder a half inch behind the vesical orifice of the urethra, and sutured the mucous membrane of the bladder to the vaginal mucosa, thus forming a vesico-vaginal fistula a half inch in length. I did this in the hope that the prevention of the accumulation of any urine in the bladder, would also put an end to the vesical spasms, hoping also, that with the complete rest from pain, there would be a simultaneous general improvement. In this I was deceived, as the spasms continued in spite of the drainage. After waiting several weeks therefore, I again placed her under chloroform, denuded the margins of the artificial fistula, and closed it.

At the same sitting, I proceeded to carry out a plan by which I proposed to get at the stricture more directly, and to explore the ureter beyond it as well as the kidney, by means of an incision into the anterior vaginal wall, exposing the ureter, which was to be opened in its long axis and its lining membrane sutured to the vaginal mucosa,—*i. e. kolpo-ureterotomy.*

For this purpose, the vagina was well exposed by Sim's larger speculum, and an incision made in the anterior lateral vaginal wall 3 cm. in length, beginning just in advance of the cervix and extending in a straight line down the sulcus towards the vaginal outlet; the vagina was cut through and the cellular tissue exposed, and widely separated by the fingers. The speculum was then removed, the finger introduced, and the ureter felt through the cut, with more difficulty than through the intact vaginal wall; it was at once caught on a blunt hook grasping a large bundle of the surrounding cellular tissue. The Sim's speculum was then reintroduced and the tissues on the

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tenaculum cautiously divided with a knife. It proved so difficult to distinguish the ureter from the other bundle of white fibrous tissue on the stretch, that I injected the bladder with four ounces of a salt solution (a drachm to the pint), and with patient still in the lateral position, introduced a whalebone catheter through urethra and bladder, into the ureter, pushing it up beyond the field of operation; I then readily cut down on to the catheter, opening the ureter longitudinally. The catheter was left in place to serve as a guide, and a grooved director carried in alongside of it and pushed up in the pelvis. Upon withdrawing the grooved director, a modified Pawlik's catheter was carried up through the artificial fistula, through which there was at once a discharge of turbid urine trickling drop by drop. The bite of the catheter in the stricture of the ureter a few centimeters above the opening was distinctly felt. A pair of delicate curved dilating forceps were next introduced, and the blades gently separated, dilating the stricture about 3 mm. The mucosa of the bladder was now stitched by fine silk sutures to the vaginal mucosa on either side, and the long incision in the vagina closed at each end down to the artificial ureteral orifice.

The operation from beginning to end was difficult, from the inaccessible position of structures which could not be brought down within easy reach and manipulation, as well as from the small size and extreme delicacy of the ureter, which had to be handled with the utmost care. It lasted two and a half hours. The patient quickly rallied, and felt at once much relieved. The urine from the left kidney thus drained directly from the ureter into the vagina at the cul-de-sac. When the stitches were removed there remained nothing but a small red orifice high up in the vagina to the left, 4 cm. from the external urethral orifice and just in advance of the cervix. She took chloroform about once in ten days after this for the purpose of dilating the ureteral stricture through the vagina.

Twice after this I removed small calculi from the ureteral orifice. Each time there was an accession of all the old symptoms, which were relieved as soon as the calculus was removed.

Each time the catheter was carried past the stricture it was followed by a gush of urine, amounting to as much as 30 cc., demonstrating the fact that above the stricture there was a dilatation—a hydroureter.

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The stiff metal catheter was readily carried up to explore the pelvis of the kidney, 23 cm. from the orifice in the vagina.

The Urine.

The urinary and microscopical report is very interesting, as it throws important light upon the pathological conditions.

Round ureteral cells, sometimes finely granular, containing large nuclei were found singly and in groups, indicating an inflammatory disease of the ureter—a ureteritis.

The first examination was made upon 25 cc., drawn from each kidney separately by the ureteral catheter.

Left kidney, urine lemon-yellow in color, slightly opaque. Reaction faintly alkaline. On standing a slight amount of white, finely granular sediment and mucus.

Microscope.—Amphorphous phosphates, triple phosphates, and a moderate number of epithelial cells principally from the ureter.

Right kidney, urine orange color, and more opaque. Reaction neutral. More deposit on standing, more viscid and streaked faintly reddish.

Second examination a month later.

Right kidney, 14 cc. urine drawn within three minutes by catheter from the ureter, clear lemon color, faintly acid.

Albumen negative by nitric acid, trichloracetic acid and heat.

Left kidney, 60 cc. urine by catheter from ureter, pale lemon color, cloudy with light flocculi. Reaction neutral. Sp. gr. 1006. Scattering large round and smaller cells from ureter. Few leucocytes. Casts negative.

Third examination.

Right kidney, 12 cc.

Left kidney, 18 cc. drawn from ureter in three minutes.

Both specimens show a trace of albumen; less marked in that from right side. Also large round and small cells from ureter in both.

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XV.

RECORD OF DEATHS FOLLOWING GYNECOLOGICAL OPERATIONS.

By HOWARD A. KELLY, M. D.

ABSTRACTED FROM THE RECORDS OF THE PATHOLOGICAL DEPARTMENT.

The following is a record of the autopsies of all cases dying in the gynecological department after operation.

Autopsy, XXXV.

Mrs. Y. (Art. VIII, No. 396) died five days after vaginal hysterectomy. She apparently did very well until the fourth day when the pulse suddenly ran up and she began to vomit. The wound was perfectly sweet, and there was no apparent cause for her condition. I opened the vaginal wound which discharged a little sanious fluid. No satisfactory cause for death could be discovered at the post-mortem examination.

Records of Pathological Laboratory. Service of Dr. Kelly. Autopsy, 10 a. m., 11–11–1889. E. C. Y., aet. 53.

Anatomical Diagnosis.

Operation wound. Removal of ovaries and tubes.

Opening from peritoneum into vagina; fatty degeneration of the liver and kidneys. Septicaemia.

Exterior.

Body, large, well nourished, 157 cm. long. Over the anterior surface of both legs and both arms several dark discolorations are seen, with whitish centres, evidently due to hypodermic punctures. On the anterior surface of abdomen, a little to the left of the median line, 9½ cm. below the navel, is a linear cicatrix 4 cm. long, extending directly towards the pubes. Posterior surface of body, congested.

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On opening abdominal cavity the omentum is found to be adherent to loops of small intestines, somewhat congested in places. In the peritoneal cavity about 25 cc. of reddish, slightly cloudy fluid. Several coils of small intestines project into the pelvis, and one is lightly adherent to a wound surface at the bottom of pelvic cavity. At this point the intestine is covered by a layer of greyish fibrinous exudation, not easily scraped off.

A thin band of connective tissue passes from the anterior abdominal wall to the sigmoid flexure of the rectum. At this point is a stiff suture. In the cavity of the pelvis is about 25 cc. of fluid, of a dark, reddish color, cloudy, its surface covered with an oily looking thin pellicle, having cadaverous odor.

Thoracic Cavity.

Diaphragm on right side at the level of the fourth rib; on the left at the fifth rib. Left lung is free from adhesions. Right lung, adherent by firm, old adhesions.

Heart.

Both layers of pericardium are smooth. In the pericardial cavity, about 20 cc. of slightly turbid, oily looking fluid. On the auriculo-ventricular surfaces of pericardium of left ventricle, a few small ecchymoses. Heart is flaccid. In the right ventricle is a small amount of fluid blood, dark color, mixed with dark clots, of soft consistence. Same character of contents in other cavities of heart. Heart's flesh, dark and flabby. Left ventricle 14 mm. in thickness of wall; right ventricle 4 mm. Valves normal. Coronary arteries, soft. Weight of heart, 243 grams.

Lungs.

Left lung: pleural surface, smooth; on costal pleura a few ecchymoses; in the apex of the lung, a thickening and slight contraction, immediately beneath pleura.

Right lung: firmly adherent by old adhesions; its tissue more resistant than left lung. On section,—upper lobe, oedematous, mucous membrane of bronchi pale.

Liver.

Small, flaccid, yellowish-red in appearance and slightly mottled; lobules, visible. Gall bladder contains small amount of dark fluid

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bile, which is easily squeezed into duodenum. Lower portion of both lobes deeply furrowed. Measurements, 28 x 20 x 6 cm. Weight, 1050 grams.

Spleen.

Spleen's capsule shrunken; parenchyma, dark; trabeculae, visible. Measures, 10 x 5½ x 2 cm. Weight, 70 grams. Volume, 62 cc.

Kidneys.

Kidneys are flaccid; somewhat pale; capsules easily stripped off; cortex 5–7 mm. in thickness, striation normal.

Supra-Renal Capsules.—Normal.

Intestines.

Stomach and intestines normal.

Pelvic Contents.

At the bottom of pelvis is a wound opening into the vagina; the edges of which are dark, congested, and covered with a fibrinous exudation. Uterus, ovaries and Fallopian tubes, absent. The peritoneum around wound, dark and congested. In the posterior surface of the vagina, near the outlet are several incisions, each about 1½ cm. long, extending through the mucous membrane. On the anterior surface of the vagina, just beneath the wound, an incision through the mucous membrane, 8 cm. long, with separation of the edges a distance of 8 mm.

Brain.—Normal.

Microscopic Examination.

Liver;—marked fatty degeneration. In kidneys, fatty degeneration of epithelium of convoluted tubules.

Autopsy, XLVIII.

This patient (No. 27 in the laparotomy list) died ten days after an operation for removal of diseased ovaries and tubes and a fibromyoma of the fundus of the uterus. She had a secondary hemorrhage from the tube on the fifth day, following an attack of vomiting. After this her general condition was excellent up to the day before death when the pulse ran up to 150. No satisfactory cause of death could be discovered.

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Dr. Kelly's service. Pathological Laboratory Records, 12–6–1889. S. R., African, aet. 33 years.

Anatomical Diagnosis.

Operation wound. Removal of ovaries and tubes.

Exterior.

Body 157 cm. in length; strongly built and well nourished. No rigor mortis is present; on the right buttock is a small cicatrix. In the anterior abdominal wall, in the median line, commencing at a point 9 cm. below the umbilicus, is a linear wound, 4½ cm. in length; in the upper portion of which is an opening, 12 mm. in diameter, leading into the peritoneal cavity. Edges of wound, partially free from epidermis. On the left mamma, near the sternum, and on the inner surface of right arm are ecchymoses from hypodermic injections.

Peritoneal Cavity.

The subcutaneous fat is well developed; muscles red.

Peritoneum red and injected; surface smooth; mesenteric glands not enlarged; a number of coils of small intestines in pelvis adherent to one another by firm adhesions; no fluid in the pelvic cavity; liver and spleen, free from adhesions.

Thoracic Cavity.

Diaphragm on the right side in the third intercostal space; on the left, at the fourth. Pleural cavities dry.

Pericardium.

Both layers of the pericardium smooth.

Heart.

Left ventricle firmly contracted; right, flaccid; in the right side of heart, fluid blood with a few small, fresh coagula; right ventricle somewhat dilated and flaccid. Left ventricle, firm. Heart's flesh, dark red in color. Wall of left ventricle measures 14 mm. in thickness; right, 2 mm. Valves, normal. Aorta, just above valves measures 63 mm. Right coronary artery is given off high up in aorta. Coronary arteries small and normal.

Lungs.

Left lung: normal. In bronchi of right lung is a small amount of sero-purulent fluid.

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Liver.

Bile duct, open. Portal vein free. Liver large; left lobe very long, thin, and on its lower border a few old connective tissue adhesions. Gall bladder contains about 50 cc. dark fluid bile. Liver on section is dark red, smooth.

Spleen.

Spleen measures 10 x 6½ x 3 cm., firm, smooth, and on section trabeculae are visible.

Kidneys.

Left kidney,—capsule easily stripped off; surface smooth, and showing some remains of foetal lobulations. On section pale, cortex 7 mm. in thickness.

Right kidney, cortex 5 mm. Pelvis slightly dilated; capsule, non-adherent, smooth.

Intestines.

Caecum and vermiform appendix, bound down by firm adhesions. The caecum, vermiform appendix and a number of coils of intestine lie in the pelvis. The caecum just at the valve, and a loop of the ileum, a short distance above, are adherent to a firm mass, lying a little to the right of the body of the uterus; another loop of small intestine, or rather the mesentery near the loop, is adherent to the posterior portion of the uterus. On the left side, near the uterus, a portion of small intestine is adherent. Several loops of intestine are lying in the cavity, behind the uterus and rectum.

These adhesions are firm so that they are separated with difficulty. In the middle of the adhesions on the right side and completely shut off by them is a small cyst filled with thick tarry blood. The caecum and ascending colon are dark, the mucous membrane congested, dilated, and filled with a large amount of soft yellowish faeces.

Brain.—Normal.

Supra-Renal Capsules and Pancreas.—Normal.

Microscopic Examination.

Microscopic examination of heart's flesh normal. Culture tubes were made from the peritoneal cavity and from the kidneys and spleen with negative results.

Sections were made of the adhesions between the intestines, the

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uterus, and the stumps of the tubes. The mucous membrane of the adherent intestine shows an extensive small cell infiltration extending into the mucous membrane. The vessels are dilated and there are numerous small hemorrhages principally in the submucous tissue. The peritoneal surface is intimately adherent to a large coagulum. At the edges of this are numerous round cells and leucocytes; newly formed vessels enter it from the peritoneal coat. At the other side of the adhesions the coagulum has similar relations to the uterus and the stumps of the tubes.

Autopsy, LII.

This patient (No. 36 in the laparotomy list) died after hysteromyomectomy, opening the cavity of the uterus. The colon was bound down to the tumor by dense adhesions. The colon was widely torn and stitched. The left ovary and tube were dug out of adhesions. The pedicle was closed with interrupted silk sutures and dropped, and a drainage tube inserted. Gauze was packed in the pelvis and brought out at the lower angle of the abdominal incision. Infection here probably entered by the wound in the uterus. A number of cultures yielded staphylococci.

Dr. Kelly's service. From the Pathological Laboratory Records. 1–2–1890. E. K. B., aet. 33 years.

Anatomical Diagnosis.

Operation wound in abdominal wall and in pelvis. Peritonitis. Fatty degeneration of heart, liver and kidneys. Oedema of lungs. Old tuberculosis of lungs. Anaemia.

Exterior.

Body 156 cm. long, well built, slightly emaciated. On anterior surface of both thighs are marks of hypodermic injections. Posterior surface of body slightly congested. Slight rigor mortis.

Brain.—Normal.

Abdomen.

In the anterior abdominal wall in the median line, commencing 6 cm. below the umbilicus and extending downward 11 cm., is a linear wound closed by nine sutures. The upper portion of the wound is closed; the lower gapes slightly. On pressure along the wound a slight amount of clear fluid exudes. The edges of wound in the upper portion are adherent, dry and free from suppuration.

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Peritoneal Cavity.

On the peritoneal side of wound the peritoneum is injected and covered with a thin layer of fibrin. The intestines are distended; the peritoneum injected; here and there slight fibrinous adhesions between the loops of intestine. The intestines lying in the pelvis are adherent to the pelvic organs and to each other. In the peritoneal cavity there are 30 cc. of bloody fluid. The liver adherent to surroundings by old adhesions.

Thoracic Cavity.

Diaphragm on the right side is at the upper margin of fourth rib; on the left at the fourth intercostal space. Left lung is slightly adherent; the right more firmly. In the left pleural cavity 20 cc. of bloody fluid. The right moistened with fluid of the same character. In the pericardial cavity a few cc. of clear fluid.

Heart.

The heart is flaccid. On the surface of the left auricle there are several small ecchymoses. In both auricles and the right ventricle there are fresh coagula. Myocardium soft, flabby and of a dark brownish color. On the ventricular side of mitral valve there are a few small atheromatous patches. Valves otherwise normal. Coronary arteries and aorta slightly atheromatous.

Lungs.

Right lung. The anterior portion of the upper lobe emphysematous; the lower lobe and posterior portion of middle and upper lobes intensely oedematous. In the apex of lung beneath the pleura is a small calcareous and caseous nodule.

Left lung intensely oedematous. In the lower lobe are a few old calcareous nodules surrounded by indurated tissue.

Liver.

Liver small—the capsule stripped off in its removal. On section the cut surface is pale and smooth. In gall bladder dark fluid bile.

Spleen.

Spleen surrounded by adhesions. On section, of a light reddish color; trabeculae distinct.

Kidneys.—Normal.

Pancreas.—Pancreas and supra-renal capsules normal.

Stomach and Intestines.—The mucous membrane pale.

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Pelvic Contents.

The sigmoid flexure lies in the pelvis, and is adherent by easily broken adhesions. Behind the uterus and shut in by the adherent sigmoid flexure is a space filled with dark coagula. The bladder contains a small amount of cloudy urine. Between its posterior wall and the uterus is a cavity 5 cm. in diameter filled with fluid blood. This cavity is closed in superiorly by sutures between the bladder and uterus. The mucous membrane of the uterus congested and covered with a slight amount of fibrin.

Microscopic Examination.

All of the organs anaemic.

On microscopic examination, a fatty degeneration of the heart and liver is found. Slight fatty degeneration of the kidneys.

Cultures were made from the inner surface of abdominal wound, from the wound in pelvis and from the blood cyst, between bladder and uterus. In all of these pus organisms developed. Two tubes from abdominal cavity remained sterile. The richest cultures were from the blood cyst and from the space behind the uterus, from which the tumor was extracted.

AUTOPSY, LV.

Hysteromyomectomy for a large fibroid tumor filling the abdomen. Death on the fourth day from volvulus. Serious visceral lesions.

Service of Dr. Kelly. From the Pathological Laboratory Records, 1–3–1890. M. McC., aet. 46 years.

Anatomical Diagnosis.

Peritonitis. Operation. Abdominal wall, wound of the uterus and uterine appendages; volvulus of ileum; abnormally long mesentery; emphysema, oedema of lungs; slight pleurisy on left side, and slight morbus Brightii, fatty degeneration of heart and kidneys.

Exterior.

Body large, 167 cm. long, strongly built and well nourished; posterior surface congested; face slightly icteric; rigor mortis in both extremities; pupils slightly dilated; abdomen distended. On the anterior abdominal wall in the median line, commencing 10 cm. above the navel, extending downwards 21 cm., is a linear wound closed by numerous sutures with their ends projecting. At the lower angle of wound the sutures cease, and from this point a number of large, thick ligatures project. On each side of wound slight

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discoloration of the skin, the edges of the wound in most places grown together.

Peritoneal Cavity.

Subcutaneous fat well developed, yellow in color; muscles red. The peritoneum around the wound reddened, and covered by a very thin fibrinous exudation. The peritoneal edges of the wound closed, the omentum adherent to its lower portion. The peritoneum over the intestines congested and covered with a thin layer of fibrinous exudation, the different coils lightly adherent; here and there between the coils small flakes of pus. The small intestines enormously distended. In the peritoneal cavity about 50 cc. of hemorrhagic exudation. Liver and spleen free from adhesions.

Thoracic Cavity.

Diaphragm on the right side, at upper border of fourth rib; on left at fourth intercostal space. Anterior borders of lungs in contact. Right lung free from adhesions. Posterior portion of left bound down by old, easily broken adhesions. Pleural cavities, dry. The two layers of pericardium smooth; in pericardial cavity, a few cc. of slightly cloudy fluid.

Heart.

Heart in diastole, its flesh soft. In both sides of heart fluid blood and fresh, partially decolorized, clots. Both sides of heart dilated. Entire heart exceedingly soft and flabby. Slight atheroma at commencement of the aorta and on the upper ventricular aspect of mitral valve. Coronary arteries slightly atheromatous.

Lungs.

Left lung, the upper lobe and the entire anterior portion of whole lung, emphysematous; the pleural surface of lower lobe covered with numerous small ecchymoses, here and there on the surface, especially over more congested areas, a slight fibrinous exudation; lower lobe of lung intensely congested and oedematous. Mucous membrane of bronchi reddened. Right lung; anterior border and upper lobe, emphysematous. Lower lobe congested, and intensely oedematous; bronchi congested.

Liver.

Bile duct free. Liver small, surface smooth. On section, of a dark brownish color; lobules visible. Gall bladder distended with dark bile; it contains three faceted gall stones, yellowish in color.

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Spleen.

Spleen soft, its tissue smooth, relaxed; trabeculae visible.

Kidneys.

Left kidney: capsule in places lightly adherent, markings normal; upper borders of pyramids congested; cortex, 6½ mm. Weight, 130 grams.

Right kidney: of same general appearance and a little smaller than the left.

Intestines.

The upper portion of small intestine enormously distended and hyperaemic. The lower portion, consisting of almost the entire ileum, small, twisted around the abnormally long mesentery and lying in the pelvis; the overlapping portions of mesentery covered with fibrinous and hemorrhagic exudation, and lightly adherent. The vermiform appendix is 13 cm. in length, lies loosely in the peritoneal cavity and is attached to a long mesenteric process springing from the meso-caecum. This mesentery covered with slight ecchymoses and exudations.

Abdominal Wound.

The examination of the abdominal wound shows no foci of suppuration. Throughout the greater part of its extent it is perfectly united. At the lower portion is an opening passing directly into the abdominal cavity. The cervix uteri is long and thin; the uterus cut across about 3 cm. above the cervix, the stump of this adherent to the lower end of abdominal wound, the surrounding peritoneum smooth and covered with a slight layer of exudation; on the left side of uterus a stump tied off with large sutures, on the distal side of these ligatures are large thromboses vessels; on the left side the same condition.

Pancreas.

Pancreas is normal. Supra-renal capsules, normal.

Microscopic Examination.

Microscopic examination of the heart shows most extensive and diffuse fatty degeneration; the muscular fibres easily separated and broken up; fat in tolerably large drops. Kidneys show moderate degree of fatty degeneration and some diffuse increase in connective tissue. Stick cultures in gelatine tubes made from between adherent loops of intestine developed nothing.

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Vol. II No. 5

THE

JOHNS HOPKINS HOSPITAL REPORTS

REPORT IN SURGERY, I

THE TREATMENT OF WOUNDS WITH ESPECIAL REFERENCE TO THE VALUE OF THE BLOOD CLOT IN THE MANAGEMENT OF DEAD SPACES.

BY WILLIAM S. HALSTED, M. D.

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THE TREATMENT OF WOUNDS WITH ESPECIAL REFERENCE TO THE VALUE OF THE BLOOD CLOT IN THE MANAGEMENT OF DEAD SPACES.

To drain or to obliterate with the greatest care all of the dead spaces of a wound is still an almost universally accepted precept of surgery; and surgeons have a wholesome fear of the presence of blood in wounds.

Having referred to the attempts of Maas, Neuber and others to dispense with the use of drainage tubes, Küster[1] expressed himself on the subject of blood in wounds as follows: "Moreover, it soon became evident that the presence of blood in any wound represented a great danger, and after a while one seldom heard of any such experiments"—experiments to close a wound without providing for its drainage or for the obliteration of its dead spaces.

Von Bergman,[2] in an address which he delivered on the antiseptic treatment of wounds a few years ago, says: "That organic material which with the greatest ease becomes the seat and essential part of putrefactive processes is the blood, which streams or trickles from the

[1] E. Küster, Ueber die Anwendung versenkter Nähte. Archiv. für Klin. Chirurgie, 1884, Bd. xxxi, Heft 1, S. 133.

[2] Schede, Ueber die Heilung von Wunden unter dem feuchten Blutschorf. Verhandlungen der deutschen Gesellschaft für Chirurgie, 1886, S. 65.

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divided vessels of a wound and collects in its deeper parts, particularly in its recesses and pockets. Since it is the business of the surgeon to avoid doing anything which could give rise to decomposition in a wound, it should be his care to provide for the exclusion of a fluid so prone to decompose, one of the most important if not the most important of the achievements of antiseptic surgery." . . . .

"Formerly, so long as one regarded the blood as the plastic material which fills the holes, as the glue and cement which should stick together the edges of the wound, its presence seemed not only good but desirable—now, since one knows that the most feared and most dangerous wound diseases arise from its decomposition, one struggles with all one's might to keep the wound freed from it. The surgeon who has not arrested the bleeding with the most scrupulous care, will in vain look for results, for success with his antiseptic technique."

Lister has done more than any one perhaps to perpetuate this great fear—the fear of blood in wounds—of prae-antiseptic times. For twenty-three years the eyes of every surgeon have been turned towards Lister, who has rarely lost an opportunity to emphasize the importance of wound drainage. His efforts are still directed to the best means of taking care of the discharges from wounds—to the perfection of a dressing. In 1875[1] he expressed himself on the subject of drainage as follows: "Two days later, or six days after the operation, the pain in the limb, of which he had complained at the last dressing, had left him; but a considerable serous stain being still found on the gauze, the drainage-tube was continued, though shortened by cutting off a piece from the deeper end."

Two days later: "The wound being dressed the line of incision was found entirely healed, except at the point occupied by the drainage tube, and the serous stain on the dressing was so much diminished that I reduced the little tube to a quarter of an inch in length, and allowed three days to pass before the next dressing. On then exposing the wound, however, I was disappointed to find the serous stain on the gauze fully as great as on the last occasion, and pressure on the skin in the vicinity of the wound caused a drop of clear serum to escape. This had never been seen before and implied

[1] Lancet, 1875, p. 436.

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that the shortened drainage tube had not been answering its purpose completely, but had permitted a certain amount of serum to accumulate; and slight as this accumulation was, I knew from experience that it was enough to perpetuate serous oozing by the tension which it occasioned. I therefore substituted for the short drainage tube another of the same calibre, but twice as long—viz., half an inch, being as deep as it could be passed without violence, and dressed again in two days. The result was such as I had hoped. There was an almost entire absence of serous stain on the gauze, and nothing could be pressed out of the tube, which was now again slightly shortened. The dressing was then left untouched for four days, when the drainage tube was found lying out of the wound, having been forced out by the consolidation going on in the interior. There was almost no stain on the gauze, and nothing could be pressed out of the orifice where the tube had been. But to return to the point which this case is intended to illustrate—viz., the value of the drainage tube in the later stages of wounds. Supposing that instead of substituting a longer tube for the shorter one on the eighth day after the operation, when a little serum was found to have accumulated, I had then given up the use of the tube altogether, the possibility is that by the time of the next dressing the outlet at the integument having become partially occluded by granulation and contraction, a greater amount of serum would have been pent up in the cavity, and in course of time the additional tension so occasioned would have led to suppuration and to the opening up of the nearly cicatrised wound."

In 1866,[1] at the very beginning of his experiments with carbolic acid—at his third case—he described the organization of a small blood clot. He observed the case—a compound fracture of the leg—with an intense interest. I shall quote from his account of the case that part which relates to the organization of the blood clot.

"On the 7th of June, nearly three weeks after the accident, an observation of much interest was made. I was detaching a portion of the adherent crust from the surface of the vascular structure into which the extravasated blood beneath had been converted by the process of organization, when I exposed a little spherical cavity about as big as a pea, containing brown serum, forming a sort of

[1] Lancet, 1867, p. 328.

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pocket in the living tissues, which, when scraped with the edge of a knife, bled even at the very margin of the cavity. This appearance showed that the deeper portions of the crust itself had been converted into living tissue, for cavities during the process of aggregation, like those with clear liquid contents in a Gruyère cheese, occur in the grumous mass which results from the action of carbolic acid upon blood; and that which I had exposed had evidently been one of these, though its walls were now alive and vascular."

"Thus the blood which had been acted upon by carbolic acid, though greatly altered in physical characters, and doubtless chemically also, had not been rendered unsuitable for serving as pabulum for the growing elements of new tissue in its vicinity."

Although perfectly familiar with the organization of the blood clot it has, apparently, never occurred to Lister to make a systematic effort to utilize the blood clot and to imitate nature's method of disposing of the dead spaces in wounds.

He has taught us what can be done under the cover of antiseptics. One may maltreat the tissues to any extent—mutilate the wound during the operation in every possible way, cut off by ligatures the circulation in large masses of tissue, produce extensive areas of superficial necrosis by irrigation with antiseptic solutions, stuff the wound with gauze and drainage tubes, tear out the stuffing and with it the granulations which have grown into it, restuff, etc.—and still the wound may heal without suppuration, without septic inflammation, and in a way which is, perhaps, altogether satisfactory to the surgeon.

But now that wound infection is for many surgeons almost a thing of the past, we may ask ourselves if, after all, our wounds are ideal wounds. One naturally hesitates to attempt to give one's notion of an ideal wound. His ideal wound of to-day may not be his ideal wound of next year or even of to-morrow. I conceive an ideal wound to be one which immediately after the operation is reduced to the condition of a non-penetrating subcutaneous wound, and which is as free as this is from the dangers of infection. By a "non-penetrating" wound I mean one in which the skin is not penetrated and I use the term non-penetrating because the penetrating wounds which used to be made in tenotomy, joint and other operations were called *subcutaneous* wounds. A wound which has been irrigated with solu-

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tions of carbolic acid, corrosive sublimate, or other disinfectants labors under the disadvantage of a more or less extensive area of superficial necrosis from which the subcutaneous wound is free. The subcutaneous wound is not exposed to the dangers which attend the introduction of drainage tubes, ligatures and sutures, nor to the greatest of all dangers for the surgeon's wound, that of infection from the hands of the operator and his assistants. The drainage tube is still one of the chief features of the modern treatment of wounds despite the systematic efforts of many surgeons for the past ten or more years to dispense with its use.

Among the first to propose a method for securing the healing of wounds without drainage tubes was Kocher.[1] He recommended a secondary suture. His method was complicated, necessitated several dressings and a second operation, was annoying to the patient and exposed the wound unnecessarily often to the dangers of infection.

In 1884 there appeared from Neuber[2] a valuable contribution to the subject of the abolishment of wound drainage. He attached great importance to the complete obliteration of all the dead spaces in wounds, recommended for irrigation a sterilized 0.6 per cent. salt solution, and provided for drainage by "canalization" and by loose stitching. For the obliteration of dead spaces he had at his disposal the following means: compression from without, buried sutures, inverting stitches, flap implantations and firm coaptation of the resected ends of bones.

In the same year Küster[3] published his well known article on the employment of buried sutures.

Impressed by the work of Küster, Neuber and others and entertaining with them the same fears of blood in wounds, it was my practice for several years to attempt with the utmost care to obliterate the dead spaces in wounds. The results were gratifying but the technique was often very tedious. For example, after an amputation of the thigh it would not infrequently take an hour to obliterate

[1] Ueber die einfachsten Mittel zur Erzielung einer Wundheilung durch Verklebung ohne Drainröhren. Th. Kocher. Volkmann's Sammlung Klin. Vorträge. No. 224.

[2] Vorschläge zur Beseitigung der Drainage für alle frischen Wunden. G. Neuber. Kiel, 1884.

[3] E. Küster, loc. cit.

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thoroughly all the dead spaces. The mechanical problems were sometimes quite difficult, and one was perpetually annoyed by the fear that he might strangulate the tissues included in the sutures. After a time I became convinced that it was impossible to obliterate thoroughly all the dead spaces in some wounds, and I observed that wounds in which the dead spaces were not obliterated healed throughout by first intention just as regularly as did the other wounds. I was therefore quite prepared to welcome Schede's article on the healing of wounds under the moist blood scab. This contribution of Schede's[1] I believe to be the greatest which has been made to the technique of surgery since the introduction of antiseptic methods by Lister.

That Schede was impressed by the importance of the communication which he has made one may infer from his introduction.

He says: "I am fully aware that it is a thankless task to come once more before this august body to advocate a new method of treating wounds, and surely I should not have the courage to do so if I did not believe that what I have to communicate to you to-day deserves because of its essential importance to occupy a higher plane than the ordinary modifications of the antiseptic wound dressing, and if I were not firmly convinced that you will recognize in what I have to say a real contribution to our science. . . . It has required but a short experience with antiseptic surgery to enable us with astonishment to recognize that even very large blood clots in open wounds do not necessarily undergo destruction and decomposition, that they do not necessarily give rise to inflammation and accidental diseases, but that they can take on changes which one may designate as 'organization of the blood clot' and which one can compare with the conversion of the thrombus in ligated blood vessels."

The immortal John Hunter was many years in advance of his times when he expressed himself on scabbing and the organization of the blood clot as follows: "In many deep-seated wounds, where all the parts have remained in contact, those underneath will unite much better if the surface be allowed to scab. Some compound fractures (more especially where the external wound is very small) should be

[1] M. Schede, Ueber die Heilung von Wunden unter den feuchten Blutschorf. Verhandlungen der deutschen Gesellschaft für Chirurgie, 1886.

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allowed to heal in the same way; for by permitting the blood to scab upon the wound, either by itself or when soaked in lint, the parts underneath will unite, the blood under the scab will become vascular, and the union will be complete even when the parts are not in contact. . . . Many wounds ought to be allowed to scab in which this process is now prevented; and this arises, I believe, from the conceit of surgeons who think themselves possessed of powers superior to nature, and therefore have introduced the practice of making sores of all wounds."

In 1832 Wardrop[1] reported a remarkable case of healing under a scab. The wounded surface was, he said, the largest that he had ever beheld. It arose from the ablation of a diseased breast. The wound healed under a crust of blood which was not disturbed for thirty days.

Volkmann[2] has observed the blood clot remain in the clefts of bone for six weeks and more without becoming fluid, without undergoing decomposition and without causing any wound disturbance, and advises that it be left undisturbed. We are indebted to him for the most classical description of the macroscopic changes which take place in the blood clot of an aseptic open wound. He depicts the clot as black and glistening and apparently unchanged for six or eight days, then as consumed by granulations or gradually shrinking and changing color from black to a leather and then to an orange tint and forming at last a moist scab which drops off when cicatrization is complete. But Schede was the first to take advantage systematically of the little understood properties of the blood clot. He regarded the blood as merely a plastic material possessed of high powers of organization. From experiments of Nuttall, Prudden, Buchner, Lubarsch, Stern and others we know that the blood possesses disinfectant properties toward certain species of bacteria. Human blood serum does not appear to be injurious to the multiplication of the staphylococci and streptococci of suppuration, so that we cannot attribute the beneficial results obtained by healing under the blood clot to any direct disinfectant properties of the blood upon the pyogenic micrococci, but such properties may come into consideration in the prevention of some other forms of wound infection.

[1] Lancet, 1832–3, II, 653.

[2] Beiträge zur Chirurgie.

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SUMMARY OF ALL OF THE WOUNDS TREATED WITHOUT DRAINAGE AND WITHOUT DEAD SPACE OBLITERATION—"BLOOD CLOT WOUNDS"—

FROM THE OPENING OF THE HOSPITAL IN MAY, 1889, TO JUNE 1st, 1890.

I.	Unclassified Operations,	25
II.	Operations for Tuberculosis of Bones and Joints,	10
III.	Excision of Tuberculous Lymphomata,	10
IV.	Operations for Carcinoma of the Breast,	13
V.	Operations for the Radical Cure of Inguinal Hernia in the Male,	8
VI.	Amputations of the Thigh,	2
VII.	Arthrodesis for Paralytic Flail-Joints,	5
VIII.	Trendelenburg-Hahn Operation for Flat Foot,	2
IX.	Operations for Ununited Fractures,	2
X.	Operations for Fractures of the Patella,	2
XI.	Osteotomy for Bow Legs,	3
XII.	Incision and Irrigation of Joints for Gonorrhoeal Arthritis,	3
XIII.	Extirpation of Inguinal Glands for Gonorrhoeal Adenitis,	6
XIV.	Operations for Syphilis of Bones,	2
XV.	Necrotomies and Operations for Bone Abscesses,	11
XVI.	Extirpation of Varicose Veins of the Leg and Thigh,	4
XVII.	Operations for the Removal of Cysts and New Growths,	14
		122

NOTE.—There were no deaths. Of the clean wounds, two suppurated primarily; *vid.* p. 271.

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I.

Unclassified Operations.

Twenty-five cases. Nos. 2, 76, 78, 98, 105, 145, 160, 226, 241, 243, 284, 314, 346, 360, 377, 398, 421, 426, 445, 451, 470, 473, 479, 483, and 484.

Typical healing in 22 of the cases. Primary suppuration in two cases, Nos. 105 and 160. Hemorrhage in one case, No. 473, which necessitated the opening up of the wound.

Example 1.—*Exsection of an Elbow which had been Crushed by the Wheels of a Horse Car.* Typical healing.

Case 398. Dominick Cassine, colored, aet. 45, was admitted to the Hospital March 17th, 1890, within thirty minutes of the time of the accident. He had fallen from the front platform of a horse car. At least one of the wheels of the car had passed over his left elbow. The injury sustained was about as great as is possible in such an accident. The elbow felt like a bag of bones. The skin was broken through in three places and was badly contused on the arm and forearm as well as about the elbow.

Operation.—The existing wounds were enlarged; lacerated pieces of the triceps muscle, fragments of the lower end of the humerus and the ôlecranon process of the ulna were removed. Three or four longitudinal incisions were made through the skin which was undermined in all directions. The oozing of blood, which was considerable, was purposely not checked. The entire upper extremity was placed first in a solution of corrosive sublimate (1–1000) for about three minutes, and then in a solution of carbolic acid (1–30) for about five minutes. The wound was then allowed to fill with blood. No stitches were taken. The arm, the elbow, and the upper part of the forearm were wrapped with strips of protective about one inch wide. Iodoformized gauze was placed over the protective, and over all a very large dressing of sterilized gauze. The arm was put up in an extended position. The blood clot which occupied the wound must have been as large as a man's fist.

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March 21. The patient's pulse and temperature have been about normal ever since the operation. The highest temperature was 38.1° C. The wound is dressed, first, because the patient complains of a little pain in his fingers, and second, to satisfy our curiosity about the condition of the blood clot. The elbow is in excellent condition. Each one of the cuts is filled with a protruding blood clot.

April 2. The wound is redressed. The organization of the blood clot seems to be complete. The protruding portions of it are bright red and bleed when gently scratched.

April 12. The patient is discharged well.

October 14. The patient presents himself at the Hospital for examination. The elbow is a remarkably good one. The patient can extend, flex, pronate and supinate the left elbow perfectly. He cannot however execute these movements, particularly extension, with quite the normal force.

EXAMPLE 2.—*Extirpation of the Head of the Femur for Ununited Intracapsular Fracture of the Neck of the Femur.* Typical healing.

No. 284. Henry Franklin, aet. 50, was admitted to the Hospital January 16, 1890. In April, 1889, patient fell from a ladder and sustained a fracture of the neck of the femur. The physician who treated him did not at first recognize the nature of the injury, and after keeping him in bed for three weeks without a splint advised him to get up and walk. This the patient was unable to do. Then the physician put on a splint of some kind and kept the patient in bed for five weeks. At present, the patient can, for a moment, bear his weight on his left leg. It gives him great pain to do so. He refers the pain to the upper part of his thigh and particularly to his knee. The pain with time has increased rather than diminished.

The patient says that he prefers to die than to live as he is and is willing to expose himself to the risks, however great, of any operation which offers to him a chance of being benefited.

Examination. The leg is adducted and rotated outwards. The real shortening is about 5 cm., the apparent shortening 8 cm., and the measured shortening (measured from the anterior superior spine of the ilium to the external malleolus) 3.5 cm. The femur rotates on

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its own long axis. Diagnosis: intracapsular fracture of the neck of the femur.

Operation.—January 17, 1890. External longitudinal incision about 22 cm. long. The great trochanter was chiseled off flush with the bottom of the digital fossa. The loose head of the femur was found in position in the acetabulum and removed. It was slightly excavated. The ligamentum teres had been ruptured and not a trace of it was to be seen on the head of the femur. The neck of the femur had been absorbed. The divided muscles were loosely stitched together by a few buried silk stitches. Inasmuch as a considerable amount of blood continued to ooze from the deeper parts of the wound I introduced a narrow strip of gauze to the bottom of the wound—into the capsule of the joint. I feared that the blood might be dammed up by the tense tissues about the joint. The wound was closed by buried skin sutures and dressed with gauze.

Jan. 18. The gauze plug is removed and the large dead space which it occupied allowed to fill with blood.

Feb. 8. The wound is dressed. It is typically healed. A Volkmann's extension apparatus is applied.

April 12. Patient is allowed to walk with crutches.

Oct. 1. Patient walks very well and without pain but still wears a Volkmann's side splint. He is greatly pleased with the result of the operation.

EXAMPLE 3.—*Operation for the Cure of Deformity Resulting from a Subluxation and Fracture of the Internal Cuneiform Bone.* Typical healing.

No. 426. Miss —— was admitted to the Hospital April 15th, 1890. Eighteen months ago the horse which she was riding fell and rolled upon her left foot which was caught in the stirrup. The surgeon in attendance reduced more or less completely a dislocation of some kind which had caused a deformity conspicuous enough to attract the attention of the patient.

Ten days after the accident, while attempting to walk on crutches, the patient slipped and fell and reproduced the original deformity. The dislocation was again reduced, and a few days later again by accident produced and once more reduced. In about four months

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patient began to walk, and in a little more than a year was able to dance. About three months ago the pain in her foot, which for a time had almost vanished, returned, and since its return has steadily increased. Now it is severe enough to incapacitate her from walking.

Examination. The internal cuneiform bone is subluxated inwards and much enlarged. The foot has lost its arch and is abducted at the scaphoid-cuneiform articulation.

Operation.—The internal cuneiform bone was fully exposed by a long internal longitudinal incision. It was found to have been fractured as well as subluxated. The fracture had united with the formation of considerable callus. The internal cuneiform bone with the exception of a small disc which included the anterior surface of the bone was chiseled out. It was necessary to exert a great deal of force to restore the foot to its proper position. The cavity resulting from the extirpation of the internal cuneiform bone was allowed to fill with blood. The wound was closed with buried skin sutures, covered with gutta-percha tissue and dressed with gauze. The foot was maintained in the equino-varus position by a plaster of Paris splint. The small disc of the internal cuneiform bone was left with the hope that from it the blood clot might be converted into bone.

April 18. The wound is dressed and has united perfectly. The blood clot seems quite firm. A new and close fitting plaster of Paris splint is applied.

May 18. The foot seems to be in perfect position. The blood clot feels already as hard as bone. The patient is permitted to walk on crutches.

Nov. 1. The position of the foot is still perfect. The patient can walk many miles without the slightest discomfort. She believes that the right foot is as strong and as perfect as the left one.

EXAMPLE 4.—*Operation for the Cure of Pronation and Drop Wrist, the Result of a Fracture of the Radius and Ulna.* Typical healing.

No. 484. Conrad Pilgrim, aet. 18, was admitted to the Hospital May 27, 1890. Four months ago patient broke both bones of his left forearm about 8 cm. above the wrist. When the splints were removed patient was confident that his arm had not been properly

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set. He has not been able to extend the wrist or do any but the lightest work since the accident.

Examination. Patient's left forearm is strongly pronated and cannot be supinated. He carries his hand in the drop wrist position but is able to extend his wrist feebly. I have several times noticed this tendency to drop wrist in fractures of the forearm which have united in the position of extreme pronation. The forearm being pronated the hand must drop unless held up voluntarily by the patient. The patient soon wearies of the constant effort and the hand falls.

Operation.—May 30, 1890. Lateral incisions, 10 cm. long, over radius and ulna. The bones which were not adherent to each other were chiseled through at about the junction of their middle and lower thirds. The lower fragments were rotated outwards with considerable force. The muscles which resisted the outward rotation were necessarily twisted. The wound was stitched with buried skin sutures, covered with gutta-percha tissue and dressed with gauze. The forearm was flexed on the arm and the extreme supination maintained by a plaster of Paris splint.

June 10. The dressing is removed. The wound is healed throughout. The arm and forearm are again put up in a plaster of Paris splint.

October, 1890. Patient presents himself for inspection. He can pronate and supinate his forearm very well; can extend his wrist as forcibly as ever, he thinks, and is able to do hard work of all kinds.

EXAMPLE 5.—*Subtrochanteric Osteotomy and Tenotomies for the Cure of Deformity Resulting from Tuberculous Hip-Joint Disease.* Typical healing.

No. 445. Charles Love, aet. 10, was admitted to the Hospital May 1, 1890. Five years ago the patient began to complain of pain in his right hip and right knee. The hip soon began to swell. An abcess formed quite rapidly and opened spontaneously in several places. The sinuses persisted for two years and then closed.

Examination. The right thigh is adducted, rotated in and flexed at about a right angle to the body. The hip is not dislocated. The anchylosis at the hip-joint is apparently a bony one.

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Operation.—May 7, 1890. Open incision of the adductor muscles and of the tensor vaginæ femoris muscle. Subtrochanteric osteotomy. The thigh could then be extended and abducted. The wounds were loosely closed by buried skin sutures, covered with gutta-percha tissue and dressed with gauze. The thigh was put up in a plaster of Paris splint in an extended and strongly abducted position. We ordinarily abduct the thigh until there is apparent lengthening of the abducted limb.

May 31. The original dressing is removed. All the wounds are perfectly healed.

EXAMPLE 6.—*Exsection of a Portion of the Radius and of the Ulna for Contraction of the Flexor Muscles of the Forearm.* Typical healing.

No. 377. Clara Albert, aet. 30, was admitted to the Hospital March 17, 1890. When about one year old the patient's right little finger was crushed in the cog-wheels of a sewing machine. The wound became much inflamed and was an open sore for six or more weeks. In about five years the right hand had become strongly flexed upon the forearm. Two years later the patient was operated upon and was compelled to wear splints for three or four years. In a short time the hand became flexed again and has remained so ever since.

Operation.—March 20, 1890. A transverse dorsal incision about 4 cm. above the wrist joint was made through the skin and extensor tendons down to the bone. About 3 cm. of the radius, of the ulna, of the extensor tendons and of the skin were excised. The bones were sewed together with strong silk. The skin wound was united by buried skin sutures of fine silk, covered with gutta-percha tissue and dressed with sterilized gauze. The arm, forearm and hand were encased in the plaster of Paris splint.

April 7th. The wound is dressed. It has healed in the typical way. The patient can already flex and extend her fingers.

EXAMPLE 7.—*Arthrotomy for Rupture of the Posterior Crucial Ligament.* Typical healing.

No. 346. John H. Smith, aet. 22, was admitted to the Hospital February 26, 1890. About four weeks ago the patient was sitting

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in a chair with his legs crossed and strongly flexed. Upon suddenly extending them he "felt something slip" in his left knee joint and found himself unable to fully extend it. With the assistance of a cane he could walk without pain. About two weeks ago while manipulating his knee he unexpectedly succeeded in straightening it. About one week ago from a misstep the same slipping of something in the joint and the flexion of the joint recurred.

Examination. The leg is flexed on the thigh at an angle of about 135°. There is a little fluid in the knee joint. When the leg is strongly flexed there appears to be an almost inappreciable subluxation forwards of the head of the tibia. On forced flexion the patient complains of pain which he refers to the articular surface of the outer tuberosity of the tibia. On forced extension the patient refers the pain to about the centre of the popliteal space.

Operation.—Febuary 29, 1890. The joint was freely opened by a long antero-internal incision. The incision was curved outwards at its extremities, its lower end dividing a portion of the ligamentum patellae and its upper end some of the fibres of the vastus internus muscle. The joint contained about four or five ccm. of a clear straw-colored fluid and its synovial membrane was considerably injected. The posterior crucial ligament was found to be torn away from its attachment to the outer surface of the internal condyle of the femur and to have carried with it quite a large piece of bone in three fragments. Two of these fragments were attached to the ligament. The other fragment was loose in the joint and had made for itself a depression in the cartilage covering the antero-external surface of the internal condyle of the femur. A cavity about 2.5 cm. in diameter and about .5 cm. deep, was made in the outer side of the internal condyle of the femur by the tearing away of the posterior crucial ligament. Extension of the leg had been prevented by the impaction of one of the fragments of bone between the femur and tibia at the back part of the joint. With the liberation of this fragment the joint could be extended. The posterior ligament was cut away at its tibial attachment and removed. The wound was loosely closed with buried sutures of the muscle and skin, covered with gutta-percha tissue and dressed with sterilized gauze.

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March 2. The wound is dressed. It has healed in the typical way. The patient has no pain and the leg can already be flexed about 45°.

March 23. The patient is discharged for misdemeanor.

EXAMPLE 8.—*Division of the Flexor Tendons for Contracted Fingers.* Typical healing.

No. 243. Joseph Hettinger, aet. 28, was admitted to the Hospital December 6, 1889. Sixteen months ago the base of the middle finger and the tip of the ring finger of the right hand were crushed by cog wheels. In six weeks the wounds were healed but the injured fingers were strongly flexed.

Examination. The middle and ring fingers of the right hand are flexed and cannot be straightened. The tip of the middle finger touches the palm of the hand, and the tip of the ring finger rests on the middle finger.

Operation.—December 10, 1889. A triangular flap was made with its apex about opposite the metacarpo-phalangeal joint as in Busch's operation for Dupuytren's contraction. The contraction of the fingers was found to be due to the tendons and not to the fascia. Attempts to straighten the middle finger produced a dislocation forwards of the distal end of the phalanx. The flexor tendons of the middle finger were divided and the finger was straightened. In the gap between the divided ends of the tendons was quite a deep hole. This hole over the metacarpo-phalangeal joint and the base of the first phalanx was allowed to fill with blood. The wound was left wide open and covered with gutta-purcha tissue. The finger was bound to a narrow hard rubber splint and dressed with gauze. The tendons of the ring finger were not divided, for the flexion of this finger was believed to be due chiefly to that of the middle finger.

January 6, 1890. The wound is dressed before the Hospital Medical Society. The blood clot which fills the wound to the level of the skin is almost completely organized. The centre of the blood clot is white, but in the outer zone of this decolorized and opaque portion of the clot may be seen with the naked eye, and more distinctly with the lens, the pinkish gray translucent granulation tissue.

January 20. The wound has healed in the typical way. Both the middle and ring fingers are nearly straight. The patient can

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already flex his middle finger a little. The ring finger he flexes quite well.

EXAMPLE 9.—*Excision of the Supraorbital Nerve.* No. 105. Primary suppuration of the wound.

The wound was closed with celloidin. The hemorrhage was not properly checked; and no provision having been made for the escape of the blood, the wound became distended with it and suppurated.

I am quite sure that the suppuration in this case might have been prevented if we had opened the wound enough to relieve the tension of it. We preferred to run the risk of being compelled to use the case to demonstrate the bad effects of tension in a wound. At one time we would have referred the suppuration to the blood in the wound, and not to the tension.

This is the first closed wound which has suppurated since the opening of the Hospital.

EXAMPLE 10.—*Operation for Dislocation Backwards of the Distal Row of Carpal Bones.* Suppuration of the wound.

No. 160. Jas. W. Plummer, aet. 55, was admitted to the Hospital October 6, 1890. Seventeen weeks ago he fell from a scaffold and struck on his right palm and hyper-extended wrist. He was treated by a physician for a sprain. For three or four weeks after the accident he suffered great and almost incessant pain. At present the slightest motion of the wrist is painful, and in bad weather the pain is quite constant and severe enough to deprive him of sleep.

Operation.—Oct. 7, 1889. The wrist joint was exposed by two dorsal longitudinal incisions, the one external, the other over the dislocated os magnum. The proximal row of carpal bones was subluxated forwards, far enough to allow the dislocated os magnum to articulate with the radius. The entire distal row of carpal bones was dislocated backwards and rested on the proximal row. The os magnum had been fractured. The entire carpus was excised with the exception of the trapezium and the pisiform bone. The wound was stitched and dressed as usual. For ten days the patient was perfectly comfortable, and his temperature was about normal.

Oct. 17. The patient complains of pain in the upper part of his forearm. This is relieved by cutting the bandage a little.

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Oct. 20. The patient again complains of pain. His temperature is normal. The dressing is removed. The wound is suppurating and the pus has burrowed up the forearm and down under the extensor tendons on the back of the hand. This is the second undrained wound which has suppurated since the opening of the Hospital six months ago.

II.

Operations for Tuberculosis of Bones and Joints.

10 cases. Typical healing in all of the cases.

Shoulder: 1 case, No. 356. Elbow: 1 case, No. 318. Hip: 1 case, No. 50. Knee: 3 cases, Nos. 215, 364, 487. Ankle: 4 cases, Nos. 128, 148, 191, 372.

Example 1.—*Exsection of the Head of the Humerus and Extirpation of the Capsule of the Shoulder Joint.*

No. 356. John Kalb, aet. 18.

Operation.—March 6, 1890. The shoulder joint was exposed by a long anterior vertical incision. The capsule of the joint was involved in the tuberculous process which had almost perforated it in one or two places. The head of the humerus was removed. In it were three large and many small tuberculous foci. The entire capsule was excised. The scapula was not involved. The cartilage covering its glenoid cavity was perfectly healthy. In closing the wound the skin only was sutured, very loosely and by a few interrupted buried skin sutures placed far apart.

March 17. The dressing was removed at a meeting of the Hospital Medical Society. The wound had healed primarily. Here and there between the sutures were small areas of granulation tissue. At the upper end of the wound could be seen the surface of a small blood clot.

Example 2.—*Exsection of the Elbow Joint.*

No. 318. John Maloney, aet. 17. Admitted to the Hospital February 7, 1890. Patient has had a tuberculous elbow for four years. There is now a sinus over the olecranon which communicates with the joint. There are scars of other sinuses which are now closed.

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Operation.—Feb. 15, 1890. The olecranon process was circumscribed by a parabolic incision into the joint. From the top of this incision a vertical incision about 5 cm. long was made through the triceps muscle. The sinus was first excised. To do this it was necessary to buttonhole the semi-circular flap. The cartilage of the joint had been almost completely destroyed. There were tuberculous foci in the olecranon process of the ulna and in the condyles of the humerus. The diseased bone was removed and the capsule of the joint carefully excised. The skin wound was loosely closed with buried skin sutures of fine black silk. Gutta-percha tissue was placed over the line of the sutures. The dressings were of sterilized gauze. The joint was maintained in position by a plaster of Paris splint.

March 14. The dressing was removed at a meeting of the Hospital Medical Society. Through the little circular hole of the semi-circular flap projected the well organized blood clot. The wound had healed throughout.

EXAMPLE 3.—*Partial Excision of the Knee Joint.*

No. 215. Mrs. M., aet. 42, was admitted November 13, 1889. She has had tuberculosis of the right knee for several years. Until recently she has been able to walk without crutches.

Operation.—November 27, 1889. The joint was opened by an anterior semi-elliptical incision. It was filled with lenticular "rice" bodies, and a straw-colored gelatinous exudate. The tubercular process had reduced the patella to a shell, and had completely undermined and almost destroyed the cartilage covering the ends of the bones. There was a large tuberculous focus in the head of the tibia. The capsule, the crucial ligaments and the semilunar cartilages were excised. The ends of the bones were shaved with a bone knife, and the tuberculous focus in the head of the tibia incompletely removed by a sharp spoon. The bones were held in position by strong silk sutures. The skin wound was closed by buried skin sutures of fine silk. Gutta-percha tissue was placed over the wound. The limb was enveloped in sterilized gauze dressings. Over all was applied a plaster of Paris splint.

Dec. 2. The wound was redressed because the original dressing had become soaked with urine. The wound was perfectly healed.

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Dec. 22. The dressings were removed and the extremity was put up in a plaster of Paris splint.

May 1. There is no sign of a return of the disease. The patient is free from pain and has been walking without crutches for several months.

This case illustrates what I have repeatedly observed, viz., that a tuberculous joint may be perfectly cured even when the diseased tissue has not been thoroughly removed. I am convinced that it is a mistake to suppose that one must thoroughly remove every particle of the tuberculous tissue, in order to arrest the tuberculous process in bones and joints. Surgeons should not congratulate themselves upon having removed all of the tuberculous tissue whenever there is no return of the disease. It is impossible to determine with the naked eye the limits of the disease. Of this fact any one who carefully controls his operative work with the microscope, may convince himself. I believe it is an accident of rare occurrence for a surgeon to extirpate absolutely every particle of the tuberculous tissue of a joint, and that the permanent cures which so frequently result from operations upon tuberculous joints require an explanation quite as much as the cases of peritoneal tuberculosis which have been cured by laparotomy require it.

EXAMPLE 4.—*Excision of the Ankle Joint.*

No. 372. Harry Smith, aet. 8, was admitted to the Hospital March 13, 1890. Patient has a tuberculous left ankle. Behind and a little above the external malleolus is the orifice of a sinus which communicates with the joint.

Operation.—March 21, 1890. The incision which began over the fibula a little above the point at which it becomes subcutaneous was continued along the posterior border of the external malleolus, and around the tip of the malleolus to its termination over the base of the fifth metatarsal bone, external to the insertion of the tendon of the peroneus tertius muscle. The tendons of the peroneus brevis and peroneus longus muscles were divided. The foot was then forcibly extended and supinated and the opposing fasciae and ligaments divided. The supination, extension and adduction were continued until the joint was dislocated. The anterior ligaments were divided from within outwards. The joint was filled with pale and flabby

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tuberculous granulation tissue. The external malleolus being removed, the lining of the joint was carefully excised. The cartilage covering the lower end of the tibia had been dissected up by the tuberculous process. A small tuberculous focus in the astragalus was chiseled out. Thin lamellae of bone were sawed off from the upper surface of the astragalus and the lower end of the tibia. The divided peronei tendons were sutured. A small counter opening was made at the inner side of the joint to guard against distension of the joint with blood. The wound was loosely closed with buried skin sutures of fine silk, covered with gutta-percha tissue and dressed with sterilized gauze. The ankle and knee joints were immobilized by a plaster of Paris splint.

April 18. The dressing is removed. The wound has healed in the typical way. There is a small round granulating spot at the site of the counter opening.

III.

Excision of Tuberculous Lymphomata.

10 cases. 7 cervical: Nos. 91, 370, 389, 410, 425, 482, and 490. 2 axillary: Nos. 69 and 147. 1 inguinal: No. 327. Typical healing in all of the cases.

Example 1.—*Excision of Cervical Glands.*

No. 425. Thomas Skinner, aet. 18, was admitted to the Hospital April 17, 1890.

Operation.—April 17, 1890. The principal incision extended from the left mastoid process to the sternum over the sterno-mastoid muscle. A second incision extended from the left mastoid process, around the angle of the jaw, to the median line of the neck. The triangular flap was reflected and the anterior fibres of the sterno-mastoid muscle were divided. The glands were very extensively involved, and many of them were intimately adherent to the tissues surrounding them. Several of the glands were ruptured during the operation and their contents spilled into the wound. The internal jugular vein was stripped quite bare from one end of the wound to the other. The wound was closed with a continuous suture.

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April 23. The wound is healed and the patient discharged from the Hospital.

EXAMPLE 2.—*Excision of Axillary Glands.*

No. 69. Mary Green, aet. 24, was admitted to the Hospital July 29, 1889. For seven years patient has been annoyed with tuberculous cervical glands. These glands have repeatedly inflamed and "burst." At present the patient's neck does not annoy her. There are several large scars in the neck but no sinuses. Four or five months ago the glands of the left axilla began to enlarge. There is now in the left axilla a painful lump about as large as a lemon.

Operation.—July 30, 1889. The axillary glands were extirpated. The wound was closed in the usnal way and the fornix of the axilla carefully held up with gauze.

August 6. The wound is dressed. It has healed in the typical way.

EXAMPLE 3.—*Excision of Inguinal Glands and of a Tuberculous Ulcer of the Foot.*

No. 327. Henry S. Young, aet. 18, was admitted to the Hospital February 13, 1890. Four years ago the patient was kicked on the outer border of his left foot. The foot swelled, and in a few weeks a sore was established in the skin over the fifth metatarsal bone. After remaining open for two years, the sore became closed for three or four weeks. It then reopened and has been a running sore ever since. Two and a half years ago the glands in the left inguinal region began to enlarge and in a short time they suppurated. About six months ago one or two glands in the left popliteal space inflamed and suppurated.

Examination. In the skin over the upper and outer surface of the fifth metatarsal bone of the left foot is a sore about 3.5 cm. in diameter which presents the appearances described as characterizing what used to be known as scrofuloderma. The granulations are subcutaneous in parts of the sore. Over the subcutaneous granulations the papillae of the skin are enlarged and covered with thick crusts of epithelium. The edges of the open sore are thin and red

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and irregularly undermined. The base of the open sore gives the picture of the tuberculous ulcer of the skin. Here and there the granulations are thinly covered with epithelium.

Operations.—February 15, 1890. 1. Quite a large piece of tuberculous skin was removed from over the tuberculous inguinal glands. The glands were carefully excised. The wound was allowed to fill with blood: it was then covered with gutta-percha tissue and dressed with sterilized gauze.

2. The ulcer of the foot was excised. The wound was protected with gutta-percha tissue and dressed with sterilized gauze.

Feb. 23. The wound of the groin is dressed. It is typically healed. The blood clot is decolorized at its centre and already organized at its edges.

March 5. The foot wound is dressed. The blood clot is completely replaced by granulation tissue which is rapidly being covered with epithelium.

IV.

Operations for Carcinoma of the Breast.

13 cases. Nos. 58, 177, 216, 326, 360, 373, 381, 385, 388, 407, 408, 454, and 489. Typical healing in all of the cases.

Example.—

No. 381. Wealthy Mason, aet. 47, was admitted to the Hospital March 20, 1890.

About one year ago the patient noticed a lump no larger than a pea just external to the left nipple. The lump has gradually increased in size and is now about as large as a hen's egg. The axillary glands are large enough to be felt.

Operation.—March 21, 1890. The knife was introduced at a point from 3 cm. to 5 cm. below the middle of the clavicle and drawn outwards on to and down the arm to a point a little below the insertion of the pectoralis major muscle. The knife was then reintroduced at the starting point and the tumor circumscribed by a skin incision which gave the diseased tissues at every point a wide

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berth—a berth of at least 5 cm. Each bleeding point as it presented itself was caught at once by an artery clamp. The tumor, the entire breast and all of the healthy tissues which had been circumscribed by the skin incision were removed in one piece from within outwards, by cutting and tearing, from the ribs and from the fascia which covers the greater pectoral muscle. The triangular skin flap was dissected back to its base. The loose fascia which stretches from the lower border of the free edge of the pectoralis major muscle to the chest wall was torn through with the fingers, the major muscle was raised up from the chest wall and from the pectoralis minor muscle and cut away close to its trunk attachments and at about 5 cm. from its insertion into the humerus. The pectoralis minor muscle was divided transversely at about its middle and drawn upwards so as to completely expose the extreme apex of the axilla under the clavicle. The loose cellular tissue about the first portion of the axillary vein was dissected away with the fingers so as to clearly expose the axillary vein. Starting from this point the tissues were dissected clean from the axillary vessels and nerves, down almost to the lower limit of the skin incision on the arm. Going back again to the apex of the axilla, the axillary contents and with them all the cellular tissue and fat which covers the front and side of the exposed chest wall were dissected off clean from the ribs. The somewhat wedge-shaped contents of the axilla were thus removed in one piece from the apex to the base or floor of the axilla. The floor of the axilla had already been reflected in the triangular skin flap. The last cutting act of the operation, therefore, was to dissect the base of the wedge-shaped contents of the axilla from the reflected triangular flap of skin.

Two strong silk approximation sutures were taken from the under side of the skin at about 1.5 cm. from its cut margins. These sutures, stretched across the open wound, did not touch the ribs but were suspended in the air about midway between the ribs and the level of the skin. The flap was then pressed up into the axilla to as high a point as possible and was held there by an assistant while its edges were stitched with buried skin sutures to the skin of the chest wall. The open wound was allowed to fill with blood. The approximation sutures became completely buried in the blood clot. The

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blood clot was protected from the dressing by strips of gutta-percha tissue. The fornix of the axilla was made as high as possible and its high position was maintained by a wedge of gauze which was held in place by a firmly applied bandage. The inner dressing was of sterilized gauze and the outer dressing of cyprus moss.

April 7. The wound is dressed. It has healed in the typical way. The blood clot, which is already almost completely organized, fills the open wound up to the level of the skin. The approximation sutures are buried out of sight in the blood clot. The positions of the approximation sutures are indicated by little convexities of the skin at the margins of the blood clot: *vid.* Plate I, o, o, and ×, ×. A little above the centre of the open wound white spots may be seen in the photograph, and at the lower part of the open wound near its inner edge are some dark spots. The white spots represent the decolorized remains of the blood clot, and the dark spots represent the most deeply pigmented areas of the blood clot. The granulation tissue had reached the surface everywhere except at these light and dark spots. Sometimes at the first dressing the approximation sutures may be seen shimmering through the surface of the blood clot. They soon become entirely concealed by the granulation tissue.

About eight years ago I began not only to typically clean out the axilla in all cases of cancer of the breast but also to excise in almost every case the pectoralis major muscle, or at least a generous piece of it, and to give the tumor on all sides an exceedingly wide berth. It is impossible to determine with the naked eye whether or not the disease has extended into the pectoral muscle.

From the careful microscopical examination of many very small cancers of the breast I am convinced that the pectoralis major muscle is usually at the time of the operation involved in the new growth. Strange to say, no authority so far I know suggests the advisability of always removing the pectoralis muscle or a portion of it in operations for the cure of cancer of the breast; and still stranger there are many surgeons of the first rank—surgeons in favor of methodically cleaning out the axilla—who instead of recommending the excision of the muscle advise the removal of the *fascia* only from the pectoral muscle. Kõnig,[1] for example, in the fourth edition of his Surgery

[1] F. König, Lehrbuch der Speciellen Chirurgie, 4 ed., Vol. II, p. 107.

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says: "When the fascia over the pectoralis muscle is diseased it (the fascia) must be removed." Surely it is absurd not to remove the muscle when its fascia is, even to the naked eye, diseased.

Küster[1] in describing his method says: "Now the breast and with it the fascia pectoralis is detached from below upwards." He adopts Volkmann's technique and apparently accepts his views on the importance of removing the pectoral fascia.

Volkmann[2] in his Beiträge zur Chirurgie writes as follows: "I make it a rule never to do a partial amputation for cancer of the breast, but remove the entire breast even for the smallest tumors, and at the same time I take away a liberal piece of skin. The skin defect is, of course, very great when one operates in this manner, and the wound, in consequence, requires a long time for healing. Furthermore, in making the lower incision I cut right down to the pectoralis muscle and clean its fibres as I would for a class-room dissection, carrying the knife parallel with the muscular fasciculi and penetrating into their interstices. The fascia of the muscle is, accordingly, entirely removed. I was led to adopt this procedure because, on microscopic examination, I repeatedly found when I had not expected it that the fascia was already carcinomatous, whereas the muscle was certainly not involved. In such cases a thick layer of apparently healthy fat seperated the carcinoma from the pectoral muscle and yet the cancerous growth, in places demonstrable only with the microscope, had shot its roots along the fibrous septa down between the fat lobules and had reached and spread itself out in flat islands in the fascia. It seems to me, therefore, that the fascia serves for a time as a barrier and is able to bring to a halt the spreading growth of the carcinoma."

[1] E. Küster, Verhandlungen d. deutsch. Ges. f. Chir., 1883, p. 295.
[2] R. Volkmann, Beiträge zur Chirurgie. Leipzig, 1875, p. 329.

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V.

Operations for the Radical Cure of Inguinal Hernia in the Male.

8 cases.[1] Nos. 94, 171, 250, 329, 330, 460, 448, and 481. Typical healing in seven of the cases. In one case, No. 94, the bladder had been included in one or more of the sutures.

Example 1.—

No. 339. Henry Smith, negro, aet. 37, was admitted to the Hospital February 19, 1890. For two years the patient has had a small right oblique inguinal hernia. At present the hernia is about as large as an orange and is easily reducible.

Operation.[2]—February 20, 1890. The skin incision extended from a point about 2 cm. internal to the anterior superior spine of the ilium to the spine of the pubes; *vid.* Plate II. The subcutaneous tissues were divided so as to expose clearly the aponeurosis of the external oblique muscle, the external abdominal ring and the sac of the hernia. The aponeurosis of the external oblique muscle, the internal oblique and the transversalis muscles and the transversalis fascia were severed to the outer extremity of the skin incision. An incision large enough to admit two fingers was then made into the sac. The index and middle fingers of the left hand, and a small piece of sterilized gauze were passed into the sac. By them the hernial contents were pressed back into the abdominal cavity and over the fingers the sac, first on one side and then on the other, was drawn tense and held by the thumb of the same hand, while the tissues in which the sac was imbedded were stripped off from it by

[1] Feb. 17th, 1891. I have performed the operation, as described below, for the radical cure of inguinal hernia twenty-one times. In no case, in so far as I have been able to inform myself, has the hernia returned. Last evening, at a meeting of the Hospital Medical Society, eleven of the cases presented themselves for examination. In one case only I did not transplant the cord; in this case the hernia has returned.

[2] *Vid.* the Johns Hopkins Hospital Bulletin, 1889, Vol. 1, No. 1.

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the other hand. With the division of the abdominal muscles and transversalis facia the so-called neck of the sac vanishes. There is no longer a constriction of the sac. The communication between the sac and the abdominal cavity is more than large enough to admit one's hand. The sac having been completely isolated, was torn more widely open, and the peritoneal cavity was closed as deeply as possible by seven or eight quilted sutures of fine silk. The sac was then cut away quite close to the line of the peritoneal sutures. The vas deferens and its vessels having been isolated, they were hooked up into the outer angle of the wound by a quilted suture, which included the transversalis and internal oblique muscles and the aponeurosis of the external oblique muscle. This suture was the first of a row of seven or eight quilted sutures of strong silk, which were passed deeply through the pillars of the ring, and through the divided muscles of the abdominal wall. These sutures were taken very close together, were made to include the deepest tissues available and were tied tight enough to bring into close apposition the broad surfaces which they embraced.

Great care having been taken to ligate every bleeding point, the wound was closed with buried skin sutures of fine black silk. A pad of sterilized gauze about 3 cm. broad and a little longer than the wound was pressed over the line of the skin incision. The skin of the abdomen, thigh and scrotum was carefully dried. The pad was bound firmly in position by a few spica turns of a gauze bandage which had been soaked in absolute alcohol. Finally thin collodion was poured over the entire dressing. In a few moments the dressing was quite hard and the patient was transferred to the stretcher.

March 1. The wound is dressed. The wound is perfectly healed. Nothing but the finest linear scar is to be seen; *vid.* Plate II.

March 6. Thirteen days[1] after the operation the patient is allowed to get out of bed and to walk about.

Nov., 1890. Nine months after the operation the scar from the operation is scarcely visible. There is no return of the hernia.

[1] The patients, as a rule, are not allowed to walk about until the twenty-first day after the operation.

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EXAMPLE 2.—

No. 94. This is the only case which did not heal in the typical way. John Bleecher, aet. 48, was admitted to the Hospital August 15, 1889. Patient has an oblique inguinal hernia on both sides. The hernia on the right side is the larger and has existed for about fifteen years. It has caused him so much pain of late that he desires to be cured of it by operation.

Operation.—August 16, 1889. The operation was done in the way just described. The ring was large enough to admit four fingers.

Aug. 20. Patient has not been able since the operation to micturate without great difficulty. It is only by getting on his hands and knees that he has been able to do so. His pulse to-day is 136, and his temperature is 39.3° C. The dressings are removed. The patient is anæsthetized, and the wound, which looks as if it were healed, is cut open. There escapes from the wound a considerable amount of a very thin, brownish, somewhat blood-stained fluid. The wound is left open.

Aug. 24. Patient's temperature and pulse are still a little above normal. His dressings were found soaked with urine this morning. The urine certainly reaches the dressings by way of the wound, and hence it is probable that the wall of the bladder was caught by one or more of the deep sutures.

Sept. 8. Three days ago the patient voided some of his urine in the natural way, but until to-day the greater part of the patient's urine has escaped through the wound.

Sept. 10. There has been no urine on the dressings since the last note.

Nov., 1890. The patient made a perfect recovery, and up to date, 15 months after the operation, has had no return of his hernia.

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VI.

Amputations of the Thigh.

2 cases. Nos. 313 and 364. Typical healing in both cases.

Example.—*Amputation of the Thigh at the Lower Part of its Upper Third for Sarcoma of the Knee-Joint.*

No. 313. W. B. Griffen, aet. 22.

Operation.—April 26, 1890. Neither the tourniquet nor the Esmarch bandage was made use of. The vessels were tied as they were encountered and before they were divided. The patient did not lose more than one or two ounces of blood. The patient was in a bad condition for the operation. But the loss of blood was so little that his pulse was about as strong after the operation as it was before it. A circular and two lateral incisions were made through the skin. The rectangular flaps were dissected back about 6 cm. The vessels were divided separately. The muscles and skin flaps were intentionally made quite short. Long flaps are of course unnecessary when a wound unites by first intention. The muscles were brought together quite loosely by fine silk buried sutures. No attempt was made to obliterate the rather large dead space between the sawed end of the bone and the muscles. The danger of strangulating tissue in the effort to obliterate this dead space is so great that I prefer to allow a blood clot to occupy it. The smaller dead spaces were ignored. The skin flaps were trimmed short and their edges were brought together gently by buried skin sutures of fine silk. These sutures were placed about 3.5 cm. apart. Abundant opportunity was thus afforded for the escape of blood. The skin wound was covered with gutta-percha tissue. The dressing was of sterilized gauze. The stump was flexed at right angles to the trunk and held in place by a plaster of Paris bandage.

May 5. Nine days after the operation, the dressing was removed at a meeting of the Johns Hopkins Hospital Medical Society. The wound had healed throughout by first intention.

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VII.

Arthrodesis for Paralytic Flail-Joint.

5 cases. Nos. 178, 322, 348, 416, 444. Typical healing in all of the cases.

Example.—

No. 348. Mollie Morris, aet. 17, was admitted to the Hospital February 26, 1890. She had an attack of infantile paralysis when she was two years old. Her right ankle is now a flail-joint. She brings her foot to the ground in an extreme valgus position. Her foot pains her almost continuously whether she uses it or not. The pain is very great when she attempts to walk.

Operation.—The joint was opened by an anterior transverse incision. Everything encountered except the anterior tibial vessels and nerve was divided. The articular surfaces and with them thin discs of the tibia, fibula and astragalus were removed. The tibia and astragalus were held together by strong silk sutures. The skin wound was united by buried skin sutures, covered with gutta-percha tissue and dressed with sterilized gauze.

March 1. The dressing is removed in order that a close fitting plaster of Paris splint may be applied. The wound is perfectly healed.

April 4. The plaster splint is removed. There is firm union between the tibia and the astragalus.

Oct., 1890. The position of the foot is still good and the patient walks well and without pain.

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VIII.

Trendelenberg-Hahn Operation for the Cure of Acquired Flat Foot.

2 cases. Nos. 334 and 466. Typical healing in both cases.

Example.—

No. 466. Charles Brown, aet. 15, was admitted to the Hospital May 20, 1890. About one year ago patient's left foot began suddenly to give him pain when he walked. The foot became swollen, and when the swelling had disappeared the patient noticed that one of the "ankle bones" was projecting.

Examination. The patient's foot pronates when he walks. He walks upon the inner side of his foot. He has a typical flat foot.

Operation.—May 21, 1890. The tibia was chiseled through a little above the ankle joint as advised by Trendelenberg and Hahn. As it was still impossible to bring the foot into a strong varus position, a wedge-shaped piece of bone was removed from the tibia. Without much force the foot could then be brought into a satisfactory position. The skin wound was stitched with buried skin sutures, covered with gutta-percha tissue and dressed with sterilized gauze. The varus position was maintained by a plaster of Paris splint.

June 1, 1890. The wound is dressed. It has healed primarily. A close fitting plaster of Paris splint is applied.

IX.

Operations for Ununited Fractures.

2 cases. Typical healing in both cases.
Humerus: 1 case. No. 419. Femur: 1 case. No. 449.

Example.—

No. 449. I. A. Dorsey, aet. 54, was admitted to the Hospital May 2, 1890. In October 1889, the patient sustained a simple

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fracture of the right femur. He was treated for seven weeks with a Smith's anterior splint, and has not been able to walk since the accident.

Examination. The right thigh is 7.5 cm. shorter than the left. There is an ununited fracture of the right femur at a point a little above its middle. The upper fragment is abducted and rotated outwards. Its lower end is sharp and pointed, and has almost perforated the skin.

Operation.—May 7, 1890. The inflamed skin over the lower end of the upper fragment was excised. A longitudinal incision, about 15 cm. long, was made on the outer side of the thigh. From the middle of this incision and at right angles to it a transverse incision, about 5 cm. long, was carried towards the back of the thigh. The ends of the fragments were so greatly atrophied and overlapped so much, that it was thought advisable to remove 4 cm. from the upper and 3 cm. from the lower fragment. The bones were drilled and fastened together by strong silk. The skin wound was stitched with buried skin sutures and covered with gutta-percha tissue.

No attempt was made to obliterate by buried sutures the dead spaces of the wound. The dressings were of sterilized gauze and cyprus moss. The thigh was immobilized in a plaster of Paris splint.

May 31. The wound is examined. It has healed primarily. There is as yet no evidence of bony union.

X.

Operations for Fractures of the Patella.

2 cases. Nos. 247, 362. Typical healing in both cases.

Example.—

No. 247. Robert Sommering, aet. 41, was admitted to the Hospital December 10, 1889. A few hours before admission the patient tripped over a car track, and in falling "felt both knee caps break" before they touched the ground.

Dec. 11. The right knee was put up in an apparatus by Dr. Brockway, the house surgeon.

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Operation.—Dec. 13. A transverse incision was made into the left knee-joint between the fragments. The upper fragment did not comprise quite the whole of the upper third of the patella. This fragment was tilted in such a manner that its fractured surface looked almost directly upwards as the patient lay upon his back. All the tissues from the skin down to the bone had slipped in between the fragments; so that to bring the fragments accurately together without an operation would have been impossible. The tissues interposed between the broken surfaces were cut away and the fragments were accurately brought together by sutures which did not pass through the bone. The skin wound was united by buried skin sutures, covered by gutta-percha tissue and dressed with sterilized gauze.

Dec. 17. The wound is dressed. It has healed primarily. The left leg is put up in a plaster of Paris splint. Between the fragments of the non-sutured patella is a gap of about 1.5 cm.

June, 1890. Patient has had another fall and has torn the ligamentous union which existed between the fragments of the patella which was not sutured. He has already almost perfect motion of the left knee. The left patella is apparently perfect. A few days after the readmission of the patient, Dr. Brockway, the house surgeon, sutured the fragments of the right patella.

XI.

Osteotomy for Bow-Legs.

3 cases. Nos. 236, 336, 410. Typical healing in all of the cases.

Example.—

No. 410. Richard Schmidt, aet. 7, was admitted to the Hospital April 9, 1890. His parents state that his legs began to bend when he was two years old. The bowing has increased steadily from that time to this. The boy has now well marked anterior curvature and very exaggerated lateral curvature of both legs.

Operation.—April 10, 1890. Both legs were operated upon. Transverse skin incisions were made opposite the apices of the lateral

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curves. The tibiae were divided with the osteotome. The fibulae were broken by hand. The skin wounds were united by buried skin sutures, covered with gutta-percha tissue and dressed with gauze. The legs were immobilized in plaster of Paris dressings.

April 27. The dressings are removed. The wounds are perfectly healed. There is already considerable bony union of both tibiae. The slight deformity which still remains is corrected and the legs re-encased in plaster of Paris.

May 22. Patient's legs are firm and quite straight. He is allowed to walk.

XII.

Incision and Irrigation of Joints for Gonorrhoeal Arthritis.

3 cases. No. 77, knee; No. 104, ankle; No. 172, wrist. Typical healing in all of the cases.

Example.—

No. 77. John Schlenck, aet. 57. Admitted to Hospital August 2, 1889. Patient has gonorrhoea. His right knee and left ankle joints are distended with fluid and are very painful. There is considerable oedema of the right leg and of the left foot.

Operation.—August 3, 1889. An incision about 10 cm. long was made along the inner edge of the patella and into the joint. The joint was distended with a blood-stained, sero-purulent exudate. The fluid was evacuated and the joint irrigated for twenty minutes with a solution of corrosive sublimate (1–50,000). Two or three interrupted silk stitches were taken in the capsule of the joint. The muscle wound was not sutured. The skin wound was stitched with buried sutures of fine silk and covered with gutta-percha tissue. The knee was dressed with sterilized gauze and fixed in an extended position.

Aug. 14. The dressings are removed. The wound is healed. There is no fluid in the joint, but flexion of the joint is quite painful.

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Sept. 20. The patient can flex his knee joint perfectly and without pain. We have made several attempts to cultivate on human blood serum the gonococcus from the exudate of gonorrhoeal joints, but without success. In one instance we found, beyond a doubt, the gonococcus in the fluid from a knee joint. In every case we have made the usual test tube inoculations for pyogenic micro-organisms and in no case did a single colony develop.

XIII.

Extirpation of Inguinal Glands for Gonorrheal Adenitis.

6 cases. Nos. 344, 358, 392, 397, 432. Typical healing in five of the cases.

In the unsuccessful case, No. 344, the original dressing was removed a few days after the operation at a meeting of the Hospital Medical Society. The wound had healed primarily but the blood clot did not completely fill the uncovered dead space over the saphenous opening. The wound had probably been investigated by the fingers of members of the Society and hastily redressed. At the second dressing the blood clot was found to have broken down and there was pus at the bottom of the dead space.

Example.—*Double Inguinal Bubo.*

No. 358. John B——, aet. 20, was admitted to the Hospital March 5, 1890. The skin over the glands of the right groin is not inflamed. An incision had already been made into the glands of the left groin.

Operation.—March 6, 1890. An elliptical piece of skin which included the old incision was removed from over the glands of the left groin. Irregular T-shaped incisions were made in both groins and the glands thoroughly extirpated on both sides. Several of the softened glands burst during the operation and evacuated their contents into the wounds. It seemed advisable, therefore, to irrigate both wounds for several minutes with a solution of corrosive subli-

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mate, 1-1000. The wounds were sewed with buried skin sutures. At the bottom of each wound, over the saphenous opening, was a rather large dead space. On the left side the dead space could not be covered by skin. These spaces were allowed to fill with blood. The wounds were covered with gutta-percha tissue and dressed with gauze.

March 14. The wounds are redressed. Both wounds have healed primarily. There is a small granulating wound on the left side which represents the organized blood clot.

XIV.

Operations for Syphilis of Bones.

2 cases. Nos. 67 and 451. Typical healing in both cases.

Operation for Gumma of the External Condyle of the Humerus.

No. 67. Ida Carson, aet. 9, negress. Admitted to the Hospital July 29, 1889. Patient has hereditary syphilis. Her corneæ are opaque, her teeth are serrated. The external condyle of the left humerus is enlarged and sensitive to pressure.

Operation.—August 1, 1889. The external condyle was freely exposed by a long longitudinal incision and explored with the chisel. The surface of the exposed bone was rough and had a worm-eaten look. At about 1 cm. from the surface was a mass of necrotic tissue about the size of a large filbert. This tissue was removed and the walls of the cavity were shaved with a sharp spoon. The bone cavity was allowed to fill with a blood clot. The skin was loosely stitched with buried silk sutures. The skin wound was covered with gutta-percha tissue. The hand, forearm and arm were enveloped in a sterilized gauze dressing. The elbow was fixed in an extended position by a plaster of Paris bandage.

Sept. 10. Forty-one days after the operation, the dressing is removed for the first time. The wound is completely healed. There is perfect motion of the elbow joint. Before the operation, the elbow could not be completely extended.

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Operation for Syphilitic Periostitis of a Rib.

No. 451. Moses Conway, aet. 23, negro. Admitted to the Hospital May 5, 1890. Patient has gummata of the skull, lower jaw and rib. About 5 cm. below and 1 cm. external to the left nipple is a small fluctuating tumor. The long diameter of the swelling is parallel to that of the ribs in this situation.

Operation.—May 7, 1890. An incision, about 10 cm. long, was made into the tumor. Its soft necrotic contents were evacuated and a rib, eroded and deprived of its periosteum, was exposed. The eroded portion was removed, the walls of the cavity excised and the wound irrigated with a solution of corrosive sublimate, 1–1000. The wound was loosely closed with buried skin sutures of fine silk, covered with gutta-percha tissue and dressed with sterilized gauze.

May 10. The wound is dressed. It has healed primarily.

XV.

Necrotomies and Operations for Bone Abscesses.

11 cases. Complete organization of the blood clot and typical healing in eight cases: Nos. 142, 244, 325, 335, 366, 423, 430, and 437. Incomplete organization of the blood clot in two cases: Nos. 340 and 352. Complete disintegration of the blood clot in one case: No. 265.

Example 1.—*Sequestrotomy for Osteomyelitis of the Tibia.*

No. 437. Richard Kelly, aet. 20, was admitted to the Hospital April 19, 1890. Patient had an acute osteomyelitis of his right humerus when he was three years old, and of his right tibia when he was five years old. Both bones have been operated upon repeatedly. The sinuses of the arm and leg have healed, but there is now a large abscess over the middle of the tibia. The skin over this abscess is inflamed and very thin.

Operation.—April 23, 1890. A longitudinal incision about 18 cm. long was made over the tibia. The front wall of the involucrum

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was chiseled away. The remains of the sequestrum were removed, and the granulations lining the involucrum were most carefully scraped away. The bone cavity was thoroughly painted with pure carbolic acid. The soft walls of the abscess were excised and the entire wound was freely washed with a corrosive sublimate solution, 1–1000, and then with a carbolic acid solution, 1–20. The wound was incompletely closed by a few buried sutures. It was then allowed to fill with blood and was covered with strips of gutta-percha tissue. Moist corrosive sublimate gauze was applied next to the gutta-percha tissue, and over all a huge dressing of sterilized gauze. The knee and ankle-joints were immobilized by a plaster of Paris splint.

May 5. The dressing is removed before the Hospital Medical Society. The blood clot is organized throughout and the wound is reduced to a small granulating sore.

EXAMPLE 2.—*Sequestrotomy for Osteomyelitis of the Humerus.*

No. 423. Hermann Wunderloh, aet. 13, was admitted to the Hospital April 15, 1890. About one year ago the patient's left humerus was attacked with acute osteomyelitis. About one week after the onset of the attack an abscess appeared on the antero-internal surface of the upper part of his left arm and was lanced by the attending physician. Since then several abscesses have formed about the humerus. From all of these abscesses pieces of bone have from time to time been discharged. With the exception of one sinus at the upper part of the antero-internal surface of the arm all the sinuses are now closed.

Operation.—April 17, 1890. An incision about 22 cm. long was made from one end of the humerus to the other over its antero-internal surface. The front wall of the involucrum was freely chiseled away and two sequestra removed—one from near the upper and one from near the lower end of the humerus. The granulations lining the involucrum were carefully scraped away. The gutter-shaped cavity was thoroughly cauterized with pure carbolic acid. The wound was irrigated freely with a corrosive sublimate solution, 1-1000, and then with a carbolic acid solution, 1-20. The skin wound was loosely closed by buried skin sutures, allowed to fill with blood, covered with gutta-percha tissue and dressed with gauze.

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May 5. The dressing is removed before the Hospital Medical Society. The wound is perfectly healed.

The cases in which there was partial or complete disintegration of the blood clot—cases 340, 352 and 265—were particularly instructive ones to us. In case 340, the wound was completely filled with granulation tissue on the thirtieth day after the operation; in case 352, on the fifteenth day, and in case 265, on the twenty-sixth day.

The rapid filling of these large bone cavities, notwithstanding the fact that their blood clots had become disintegrated is, I think, to be ascribed to the treatment of the wounds. The slow filling of such cavities, when treated by the usual methods, must likewise be ascribed to the treatment. For such wounds are usually stuffed with gauze. In ten days or two weeks, the stuffing is removed, and with it the granulations which have grown into it. Then the wound is restuffed. In a few days the stuffing is again pulled out and the young granulations are again demolished. This treatment is often continued for months, and under it the granulation tissue becomes converted into dense fibrous tissue, poorly supplied with blood vessels and unable to produce anything but the feeblest granulations which finally become more or less completely covered with epithelium. And so it happens that these large bone cavities frequently do not become filled and do not heal and remain a ghastly reproach to the surgeon.

A bone cavity should never be stuffed. The granulations should be encouraged to grow as luxuriantly as posible. Whether the blood clot melts away or not, the bone cavity should be bridged over by skin or by protective or gutta-percha tissue. The granulations must be most carefully protected from insult. They should rarely if ever be irrigated. In other words, a bone cavity which has lost its blood clot, should be treated as if it still possessed it. If, after a few weeks, the cavity is not filling rapidly it should be cauterized with pure carbolic acid, its granulations should be made to bleed sufficiently to fill the cavity with blood and the wound should be given the opportunity to heal under the secondary blood clot. Suppurating wounds of soft parts should be treated in the same way. They should not be stuffed. The granulations of open sores should be protected from the dressing by gutta-percha tissue or something of this sort. In the out-patient department of the hospital opportuni-

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ties are afforded us nearly every day to observe the astonishingly rapid healing of wounds which are treated in this way. For example, a patient with scalp wounds two or three weeks old presents himself. There is considerable oedema of the scalp and the wounds which are inflamed are found stuffed with iodoformized gauze. We remove the stuffing, cauterize the wounds thoroughly with pure carbolic acid, bridge them over with gutta-percha tissue and put on a gauze dressing thick enough to protect them. If we examine the wounds within 24 hours, we find that they are filled with a more or less clear and solidified wound secretion. Within 72 hours and perhaps within 48 hours, the "secretion clot," so-called by us, is completely organized and the inflammation and oedema of the scalp have disappeared.

Example from the wards. A boy was operated upon by some one for an ununited fracture of the bones of the leg. The wound suppurated.

About two months after the operation the boy was consigned to us with a wound that was suppurating freely and which was stuffed with iodoformized gauze. We made two longitudinal incisions into the wound, removed the wire sutures, scraped away with the utmost care the granulation tissue which lined the suppurating wound and sawed off a thin piece of bone from each of the fragments. The wound was then cauterized with pure carbolic acid and loosely closed by buried skin sutures. In a few days the wound was healed.

XVI.

Extirpation of Varicose Veins of the Leg and Thigh.

4 cases. Nos. 262, 279, 296 and 328. Typical healing in all of the cases.

Example.—

No. 254. Charles H. Gaupner, aet. 48 years, has for about twenty years been annoyed by varicose veins of both thighs and legs. On admission, January 2, 1890, patient states that the pains in his legs are severe enough to incapacitate him from work.

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Examination. The long and short saphenous veins and their tributaries are dilated, thickened and very tortuous. There is oedema of the left leg.

Operation on the veins of the left leg, January 10.

An incision about 60 cm. long was made over the internal saphenous vein—the incision extended from about the junction of the lower and middle thirds of the thigh to the internal malleolus. The veins were in places (back of the internal condyle of the femur and behind the malleolus) adherent to the skin. This necessitated the making of skin flaps from the under-surface of which the veins were cut away. The internal saphenous vein was excised from one end of the incision to the other. The skin was stripped back for about two inches on both sides of the incision and the underlying veins were dissected out. The wound was closed with a continuous suture of fine black silk. No attempt was made to obliterate the dead spaces. The line of suture was covered with strips of gutta-percha tissue. The dressings were of sterilized gauze. These were not applied tight enough to interfere with the circulation in the skin, the vitality of which is impaired in operations for the removal of varicose veins: the dead spaces were, consequently, allowed to fill with blood.

Jan. 17. The wound is dressed. There is primary union throughout. The little flap behind the knee has sloughed at its edge.

Jan. 30. The little slough has become organized. Its place is occupied by granulation tissue. The patient has had no pain since the operation.

XVII.

Operations for the Removal of Cysts and New Growths.

14 cases. Nos. 82, 217, 238, 320, 354, 376, 382, 431, 433, 440, 442, 446, 471, and 491. Typical healing in all of the cases.

Example 1.—*Osteoma of the Scapula.*

No. 440. Mildred Calstron, aet. 18, was admitted to the Hospital April 29, 1890.

Operation.—April 30, 1890. The tumor, about the size of a hen's egg, was removed from the infra-spinous fossa near the axillary border

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of the scapula. The wound was loosely closed with buried sutures and the dead space allowed to fill with blood. The dead space was undoubtedly considerably obliterated by the pressure of the dressing.

May 6. The wound is dressed. It has healed primarily.

EXAMPLE 2.—*Syphilitic Testicle.*

No. 446. John H., aet. 31, was admitted to the Hospital May 1, 1890.

Operation.—May 3, 1890. Amputation of the testicle. The wound was closed with buried sutures.

May 8. The wound is dressed. It has healed primarily.

EXAMPLE 3.—*Lipoma of the Wall of the Thorax.*

No. 369. Martha Moore, aet. 40.

Operation.—March 13, 1890. Removed a flat, round lipoma about 9 cm. in diameter from the wall of the thorax. The tumor which was situated below the left breast on the prolongation of the anterior axillary line was bisected, and each half removed from within outwards. I believe this to be the easiest way to remove lipomata, cysts and other benign tumors. The wound was closed with buried skin sutures, covered with gutta-percha tissue and dressed with sterilized gauze.

March 19. The dressing is removed. The wound is typically healed. There is a very small decolorized blood clot at the inner angle of the wound.

All of the eleven cases of group XV were actively suppurating ones at the time of operation. In eight of these cases there was complete and typical organization of the blood clot; in two of them the organization of the blood clot was incomplete, and in one case there was complete disintegration of the blood clot.

If we exclude the cases of this group there remain 111 cases, 108 of which healed in the typical way. There was suppuration in two cases and hemorrhage in one case; *vid.* group I. From October, 1889, to June, 1890, all of the cases healed in the typical way.

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Of the wounds which heal primarily probably the majority do so, notwithstanding the presence of microörganisms. Success in the treatment of wounds does not depend alone upon the exclusion of pyogenic microörganisms from the wounds for the following reasons:

1. There is not, I believe, a technique which from a bacteriological point of view may be considered perfect.

2. Test tube inoculations made by us a few days after the operations from the surface of the new cicatrix, from the fine line of granulations, from the surface of the blood clot, from the tip of the gauze plug and from the bottom of the sinus occupied by the plug[1] frequently yielded a number of colonies of pyogenic organisms.

3. Injections of virulent cultures of staphylococcus aureus into the tissues and into the peritoneal cavity were not followed by suppuration nor by peritonitis.

4. Operations upon bone abscesses, the walls of which it is, perhaps, never possible to thoroughly disinfect, and upon suppurating wounds of the soft parts which likewise cannot be thoroughly disinfected were usually attended by perfect organization of the blood clot.

5. Many wounds heal primarily when no antiseptic precautions whatever have been taken.

6. The majority of subcutaneous wounds in preantiseptic times were not followed by suppuration.

7. The wounds of most surgeons heal as a rule primarily up to the drainage tubes.

Ad. 1. From the numerous experiments which we made about one year ago on disinfection of the skin we conclude that it is impossible to thoroughly disinfect it by any of the methods which have hitherto been recommended, and that it would at least consume much time to do so even occasionally by any of the means at our disposal.

In some instances we have subjected the skin to a rigorous disinfection for four days. Then test-tube inoculations were made from scrapings of the skin over the part to be operated upon. In every case we have had the dissatisfaction of finding at least three or four

[1] It was not until the Fall of 1889 that, for clean wounds, we discarded, absolutely, drainage in all of its forms. Since September, 1890, we have closed, without drainage, *all* wounds—the suppurating as well as the clean wounds.

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colonies in each of the inoculated tubes. The staphylococcus aureus was one of the prevailing organisms.

Profound constitutional effects have sometimes followed the not very prolonged [from 10 to 12 hours] application to the skin of carbolic solutions as weak as 1–60.

We have made many experiments on the disinfection of the hands. On several occasions forty-five minutes were devoted to the disinfection of one pair of hands and without success. Within the past few months Dr. Roose, one of my assistants, and Dr. Ghriskey, one of Dr. Kelly's assistants, have continued the experiments on hand disinfection under the supervision of Dr. A. C. Abbott and confirm our work of last year. The work of all who have written on the subject of hand disinfection has probably been characterized by one and the same fault. The scrapings from under the nails and from other places which have been used for test tube inoculations were perhaps saturated with the solution used for disinfection or at least contained enough of the disinfectant to inhibit the growth of microörganisms. We found that hands which had been soaked in a corrosive sublimate solution [1–1000] and were then washed with two litres of sterilized water still became quite black when immersed in a solution of ammonium sulphide. In all of our experiments, therefore, we have profited by the work of Geppert[1] and have precipitated the mercury before making the test tube inoculations. Hands which have been besmeared with a fluid culture of some microörganism can undoubtedly be so disinfected or washed so clean of the given microörganism, that no colonies of it will develop in the finger prints on a Petri's plate, and the nails of three or four fingers may in ten or fifteen minutes be so well attended to, that scrapings from the specially prepared places may be sterile. But to disinfect absolutely all parts of both hands is a different problem and one that has not been solved. In not a single instance have we succeeded in disinfecting the hands. The nearest approach to a perfect disinfection was made by the operating room nurse. On one occasion, when she had devoted nearly one hour to her hands, scrapings were made from four places. Four Esmarch tubes were made with these scrapings. Three of the tubes remained sterile. In the fourth tube there was

[1] Geppert, Berliner Klin. Wochenschr. Nos. 37 and 38, 1889.

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one colony of a long slender bacillus. Ordinarily from three to six and sometimes many more colonies appeared in each of the tubes inoculated with scrapings from hands which had been disinfected for 45 minutes. We believe that it is possible to disinfect the hands and hope that others may be stimulated by our results to investigate the subject of hand and skin disinfection afresh and with other disinfectants.

If it were possible to sterilize the hands of the operator and his assistants and the skin of the patient, the surgical technique might be made a practically perfect one. The danger of infection from the air is probably a theoretical and not a real danger. We have exposed for hours large Petri's plates—ten or twelve at a time—to the air of the operating room before, during and after operations and have never found a single colony of pyogenic microörganisms.

Ad. 2. Having assured ourselves of the fact that our technique was necessarily imperfect we became much interested in the test tube inoculations made from the surface of wounds which had healed throughout by first intention and from the bottom of sinuses occupied by the small gauze drains of wounds which had healed without suppuration. These inoculations were made at the first dressing. Not more than one-half of the tubes remained sterile. It was interesting to observe that the sinuses which at the first dressing furnished pyogenic microörganisms were often found to be perfectly healed at the second dressing. The tearing of the granulations by the withdrawal of the gauze plugs provided many of the sinuses with blood clots which became organized notwithstanding the presence of the pus-producing germs. The organization of the exudates which filled the other sinuses took place as perfectly as did that of the blood clots.

Ad. 3. We experimented[1] almost exclusively with the staphylococcus aureus, and always with cultures the virulence of which we had determined by inoculations into the ear veins of rabbits.

[1] Our bacteriological experiments were made in the Pathological Laboratory of the Johns Hopkins University.

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Inoculations of the Peritoneal Cavity with Pure Cultures of the Staphylococcus Aureus.

The peritoneal cavity was, in each instance, opened by an incision three or four cm. long through the linea alba. The wounds were closed by two rows of buried sutures and dressed with celloidin.

Group I. 2 dogs. Experiment. A small piece of potato covered with a thick growth of staphylococcus aureus [culture No. 1] was introduced into the peritoneal cavity. Both dogs died of general peritonitis.

Group II. 13 dogs. Experiment. One ccm. of a bouillon culture of staphylococcus aureus [culture No. 1] was introduced into the peritoneal cavity. Peritonitis did not develop in a single case.

Group III. 10 dogs. Experiment. One ccm. of a bouillon culture of staphylococcus aureus [culture No. 2] was introduced into the peritoneal cavity. Peritonitis did not develop in a single case.

Group IV. 5 dogs. Experiment. A small piece of sterilized potato was introduced into the peritoneal cavity. Peritonitis did not develop in a single case. The pieces of potato were usually found adherent to the great omentum and covered by a transparent and almost invisible film of connective tissue.

Group V. 7 dogs. Experiment. A small piece of potato covered with a thick growth of the staphylococcus aureus [culture No. 2] was introduced into the peritoneal cavity. All of the dogs died of general peritonitis.

Group VI. 8 dogs. Experiment. A small piece of omentum was ligated with strong silk. The ligature and the tied off portion of the omentum were inoculated with a drop or two of a bouillon culture of the staphylococcus aureus. There was fatal peritonitis in two cases, and circumscribed peritonitis in two cases. Four of the dogs recovered without peritonitis.

Injections of Pure Cultures of Staphylococcus Aureus into the Muscles.

Group I. 2 dogs. Experiment. Ligated the left femoral vein and artery about two cm. below Poupart's ligament and injected one ccm. of a bouillon culture of staphylococcus aureus into the muscles of the left leg. Within twenty-four hours there were oedema of the left leg

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and induration and tenderness at the seat of injection. Within three days the oedema had disappeared. Within a week the local tenderness and induration had also disappeared. In neither case did an abscess develop.

Group II. 7 dogs. Experiment. Ligated the right femoral artery and injected one ccm. of a bouillon culture of staphylococcus aureus into the muscles of the right leg. Within twenty-four hours there were in all cases a hard swelling and tenderness at the seat of injection. In no case was there oedema of the right leg, and in no case did an abscess develop.

Group III. 6 dogs. Experiment. Ligated the left femoral vein and injected one ccm. of a bouillon culture of the staphylococcus aureus into the muscles of the left leg. In all of the cases there supervened an oedema of the left leg and a local tenderness and swelling at the seat of the injection. In no case did an abscess develop.

Injections of the Exudate of a Purulent Peritonitis into the Skin.

2 dogs. Three ccm. were injected *into* the skin over the knees of both dogs. Only a slight reaction followed the injections.

Ad. 4. It is our practice now to treat without drainage not only clean wounds, but also almost every suppurating wound. The walls of old abscesses and sinuses are excised or, when excision is impracticable, scraped. The cutting and scraping must be done conscientiously and with great patience. The Esmarch bandage is of service in these operations. Irrigations, which we have abolished for clean wounds, may be used freely in these suppurating cases before the Esmarch bandage is removed. The suppurating wounds are carefully painted with pure carbolic acid, loosely stitched with interrupted buried skin sutures and covered with gutta-percha tissue. There is often considerable dead space in the wounds made for the cure of many and long old sinuses. The dead spaces become filled with blood after the operation. The inflamed skin surrounding the orifices of the old sinuses having been cut away, the provision for the escape of the blood from such wounds is usually more than sufficient. The dressings are very large and loosely applied. If

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the wound is between two joints it is well to immobilize both of these joints. I believe that it is particularly important to prevent a breaking up of the blood clot in suppurating cases. The technique in these cases is necessarily far from perfect. The wounds, nevertheless heal as a rule in the typical way. We must conclude that we are indebted to the tissues as well as to the antiseptics for our good results.

Ad. 5. Wounds of parts which are rich in blood vessels usually heal by first intention even when no antiseptic precautions have been taken. Operations on the face and perineum are notably successful. The pyogenic microörganisms succumb to tissues in which the circulation is so active.

Ad. 6. The majority of subcutaneous wounds of prae-antiseptic times were surely infected. Subcutaneous wounds were made with knives which had never been disinfected and which perhaps for years had been used to incise abscesses. But the subcutaneous wounds rarely suppurated. Open wounds, on the other hand, suppurated with great regularity. If an open wound were to be distinguished from a closed wound merely by the length of the incision, we might accept the generally received explanations for the suppuration of the former. I believe that the immediate cause of the suppuration is usually the rough manipulation of the infected tissues and the strangulation of the tissues by infected ligatures and sutures.

Ad. 7. Few surgeons now-a-days have a technique bad enough to prevent the union of raw surfaces which have a good circulation, if the dead spaces have been thoroughly drained and if the tissues have not been constricted by ligatures and sutures.

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THE TREATMENT OF WOUNDS.

The Management of the Dead Spaces.

Whether a surgeon disinfects by carbolic acid, corrosive sublimate or by steam; whether he uses catgut or silk for his sutures and ligatures; what material he uses for his dressings; whether the dressings are simply sterilized by heat or impregnated with antiseptics, are matters of minor importance to the patient.

Of great consequence is *The Management of the Dead Spaces in Wounds.* These may be obliterated, drained or allowed to fill with blood.

The more imperfect the technique of a surgeon the greater the necessity for drainage. The most rigid antiseptic precautions are demanded for the obliteration treatment of dead spaces: the buried sutures employed to obliterate the dead spaces necessarily enfeeble the circulation and impair the vitality of tissues which otherwise might be able to dispose of large quantities of microörganisms.

It is doubtful if one ever obliterates absolutely the dead spaces of a wound: and certainly most undrained wounds contain small blood clots however patient and conscientious and skillful the attempts at obliteration may have been. Upon the organization of small blood clots, therefore, depends more or less the healing of all undrained wounds.

But the unintended blood clot of an incompletely obliterated dead space is, I think, much more likely to decompose than the intended blood clot because it—the former—lies in tissues the circulation of which has been interfered with by buried sutures, and in a wound which is particularly exposed to the dangers of strangulation and tension.

The blood clot treatment of dead spaces has the following advantages:

1. Tissue defects are beautifully repaired.[1]
2. Obliteration sutures are dispensed with.

[1] Experiment upon a dog. We removed a piece of the triceps muscle and trephined the external condyle of the humerus. The wound was allowed to fill with blood and was covered with gutta-percha tissue. No stitches were taken. The

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Sutures and Ligatures.

I believe that the obstruction to the circulation produced by sutures and ligatures is often the immediate cause of suppuration in infected wounds, and that the larger the masses of tissue constricted and the tighter the strangulation the greater the danger of suppuration. The complete strangulation of large masses of tissue we try to avoid by using very weak silk for the sutures and ligatures.

The occasional breaking of the silk reminds one that he is tying with too much force or that he has included too much tissue in his ligature. We use the finest black sewing silk—Nos. OO, O and A. For large arteries we sometimes double the fine silk, and for deep sutures we often use the coarser numbers. Inasmuch as we cannot disinfect the skin thoroughly, we never perforate the skin with a suture. The skin wound is invariably closed by "buried skin sutures." These sutures are taken from the under-surface of the skin. They are fully described in the Bulletin of the Johns Hopkins Hospital.[1]

We consider these sutures an important contribution to the technique of surgery. The wounds are not exposed to the suppuration which so often has its origin from the sutures of the skin. It is not necessary to remove the buried skin sutures. In from three weeks to two months they appear on the surface as little loops and may be brushed away. If the sutures are well applied the scars may be almost invisible. In Plate II such a scar is represented.

The manipulation of the ligatures is a weak point in the technique of almost every surgeon. The ligatures are often cut before the operation and allowed to hang over the edge of the dish in which they are arranged; or they are brought in contact with something as they are passed from the instrument table to the operator; and even in the hands of the operator the ligatures are still in danger of becoming

extremity was immobilized in a plaster of Paris splint. In three weeks the plaster was removed. The defect in the bone was so perfectly repaired that it was impossible to see with the naked eye a line of demarcation between the old tissue and the new.

[1] These stitches do not perforate the skin, and when tied they become buried. They are taken from the under side of the skin, and made to include only its deep layers—the layers which are not occupied by sebaceous follicles. *Johns Hopkins Hospital Bulletin*, Vol. I, p. 13.

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FIG. 1.

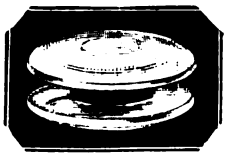
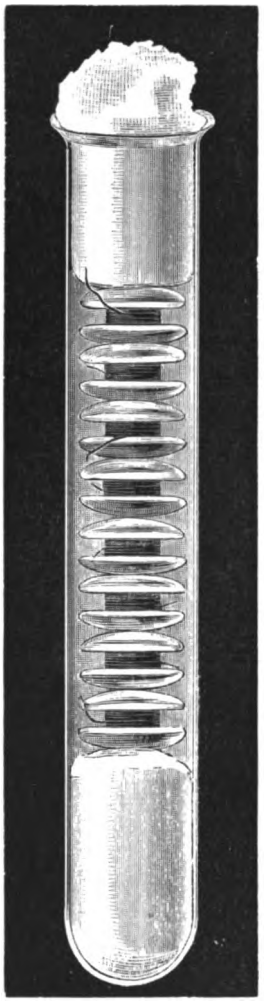


FIG. 2.



infected. When fifty ligatures, more or less, are used at an operation it may be ascribable to good luck if all of the ligatures escape infection. We conclude from our experiments that the infection of a wound from ligatures and sutures is more likely to be followed by suppuration than are the infections from other sources. The chances of accidental infection from ligatures are diminished by the employment of glass spools[1] such as are represented in Figs. 1 and 2.

A spool wound with about 2 meters of silk is held in the left hand and is unwound with the right hand of the operator as he ligates the vessels.

The sterilization and preservation of the silk is with us a very simple matter. Six or eight spools armed with silk are placed in a test tube (*vid.* Fig. 2) and sterilized by steam. Before the operation the spools are poured from the test tubes into a dish containing a solution of carbolic acid, 1–30. One of the advantages of preparing silk as we prepare it is that it may be kept sterile permanently without losing its strength. If fine silk be disinfected and preserved in any of the usual ways it soon becomes so weak as to be worthless.

The Dressings.

Between our wounds and the dressings we interpose strips of gutta-percha tissue to protect the granulations. The tearing off of

[1] These glass spools were made for me by Messrs. Warmbrunn, Quilitz and Co., No. 40 Rosenthaler-strasse, Berlin. They may now be obtained from Messrs. Eimer and Amend, New York.

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a dressing into which the granulations have grown is a barbarous act which insults both the wound and the patient. Even wounds which we wish to keep open or to tampon we protect by the gutta-percha tissue. We have found this tissue of great service in the treatment of ulcers. Until we adopted the plan of protecting every wound in this way I did not know how very seriously the cicatrization of wounds was retarded by the usual treatment of them. Gauze and tree moss are the materials which we use for our dressings. They are sterilized by steam in the Arnold steam cookers. These cookers are very convenient and satisfactory sterilizers. They are so cheap that a hospital can afford to have many of them. To each dressing we assign a cooker. When a dressing is required the cooker containing it is brought into the operating room and placed on a tripod at a convenient distance from the operating table.

The Disinfection of the Hands.

The best results were obtained when water as hot as could be endured was employed in all of the acts of the disinfection. One of my assistants could soak his hands in water at 49° C. The average hand can be held in water at 47° C. without much discomfort. We have adopted the following rules for the disinfection of our hands.

1. Scrub the hands and forearms for about ten minutes with the officinal green soap in water as hot as possible. 2. Wash off the soap in three or four basins of water as hot as possible. 3. Clean the nails most scrupulously with a sharp steel instrument. 4. Soak the hands and forearms for ten minutes in a hot corrosive sublimate solution, 1–1000. This proceeding is too severe to be employed every day. It is advisable, therefore, to so group one's cases that the "clean" ones shall be operated upon only on certain days—two days, perhaps, in one week.

The Preparation of the Patient.

About 18 hours before the operation the part to be operated upon is shaved if necessary and covered with a huge poultice of green soap. Three hours before the operation the soap is thoroughly washed off in a bath of hot water and the skin of the patient is

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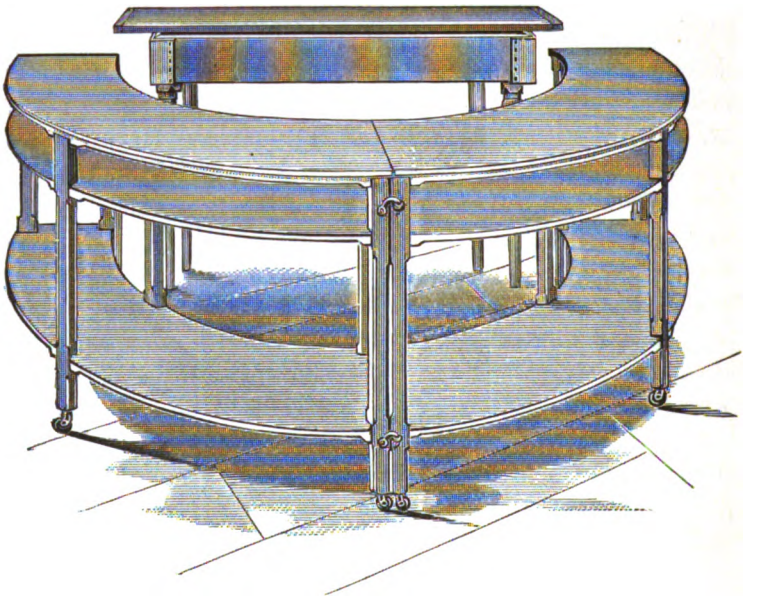
vigorously scrubbed. An astonishing amount of dessicated epithelium is scrubbed off with the soap.[1] A large wet gauze dressing is then applied. This dressing is then irrigated from time to time for an hour with a solution of carbolic acid, 1–30, and thereafter up to the time of the operation with a solution of corrosive sublimate, 1–1000. On the operating table the part to be operated upon is scrubbed and washed for a few minutes with a hot solution of corrosive sublimate, 1–1000.

FIG. 3.



The Semicircular Instrument Table and the Operating Table.

The Operation.

The operator and two of his assistants are protected from the bystanders by the semicircular instrument table (*vid.* Fig. 3). The assistant who passes the instruments wears thin rubber gloves which are disinfected in a corrosive sublimate solution. The only hands

[1] We are indebted to one of the resident surgeons of the Roosevelt Hospital, New York, for suggesting this application of the soft soap.

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which come in contact with the wound are those of the operator. The sponging is done by the operating room nurse who wrings dry the sponges, one at a time, as they are required. The pieces of gauze used for sponging are kept in a solution of corrosive sublimate, 1–1000. We consider it unsafe to use sponges which have been sterilized simply by steam unless the top of the sterilizer be removed and replaced each time that a sponge is required. It surely is not safe to expose on a table or in a basin the dry sterilized sponges to the many dangers of infection from contact.

Irrigation is employed only in suppurating cases. We have ascertained from our experiments on dogs that irrigation with solutions of corrosive sublimate as weak as 1–10,000 produces a superficial necrosis. Wounds which have been freely irrigated with solutions of corrosive sublimate not stronger than 1–5000, or moderately irrigated with solutions as strong as 1–1000, do not unite throughout by first intention. The wound in the skin unites and the divided ends of the muscles unite, but the skin is separated from the muscles by a thin serous or sero-fibrinous exudate. The cavity which contains this exudate is lined by a film of necrotic tissue thick enough to be seen with the naked eye. Little cavities filled with the same exudate are found occasionally in the connective tissue plains between the muscles.

The tissues are handled very delicately in operating. We avoid, if possible, the tearing of the tissues, and the strangulation of the tissues by ligatures and sutures. Large dead spaces are sometimes partially obliterated by buried sutures: as a rule the dead spaces are allowed to take care of themselves. The wound is closed with interrupted buried skin sutures so placed as to preclude the danger of distention. The dressing is so applied that it shall exert a gentle, even pressure. I have said that "I conceive an ideal wound to be one which immediately after the operation is reduced to the condition of a non-penetrating, subcutaneous wound, and which is as free as this is from the dangers of infection. A wound which has been irrigated with solutions of carbolic acid, corrosive sublimate or other disinfectants labors under the disadvantage of a more or less extensive area of superficial necrosis from which the subcutaneous wound is free. The subcutaneous wound is not exposed to the dangers which attend the introduction of drainage tubes, ligatures and sutures,

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nor to the greatest of all dangers for the surgeon's wound, that of infection from the hands of the operator and his assistants."

We have reduced our wounds almost to the condition of non-penetrating, subcutaneous wounds. They are neither irrigated nor drained. The chief danger from sutures is eliminated by the employment of the buried skin sutures. The strangulation of the tissues is to a great extent avoided by care in the application of the ligatures and sutures and by the use of very fine silk. The hands of the surgeon are, as a rule, the only hands which touch the wound, and these are disinfected in the manner described. The instruments are passed by the gloved hands of a trustworthy assistant; and the sponging is done by a well-trained, operating room nurse who takes them, one by one, as they are required, from the corrosive sublimate solution. I am under great obligations to Dr. A. C. Abbott for instructing in bacteriology the operating room nurse and all of my assistants, and for supervising and taking the keenest interest in our bacteriological work. All of the preparations for the operations—the recrystallization and weighing of the corrosive sublimate for the solutions, the selection and sterilization of the instruments, the preparation and sterilization of the silk and the dressings, the final disinfection of the patient—were intrusted to the operating room nurse.

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The Operating Room.

The floors, shelves and tables of the operating room are impregnated with paraffine. For the tables and shelves the paraffine is melted and rubbed in with a hot iron. For the floor the paraffine is dissolved in turpentine and painted on with a brush.

The Operating Table.

The patients are anaesthetized on long, narrow boards. They are then arranged for the operation, strapped to their boards and transferred with them to the top of a shallow sink on four legs. This sink is about 20 cm. wider and 45 cm. shorter than the board (*vid.* Fig. 4). By shifting the board the side gutters can at any moment be made as wide as may be desired. For operations on the head and feet the board is shifted in a longitudinal direction, far enough to make a gutter at the head or foot as well as at the sides of the board. The end of the board which falls within the end of the sink is supported on a small loose block which is placed on the floor of the sink. The floor of the sink drains to a hole in its centre.

FIG. 4.

The Operating Table.

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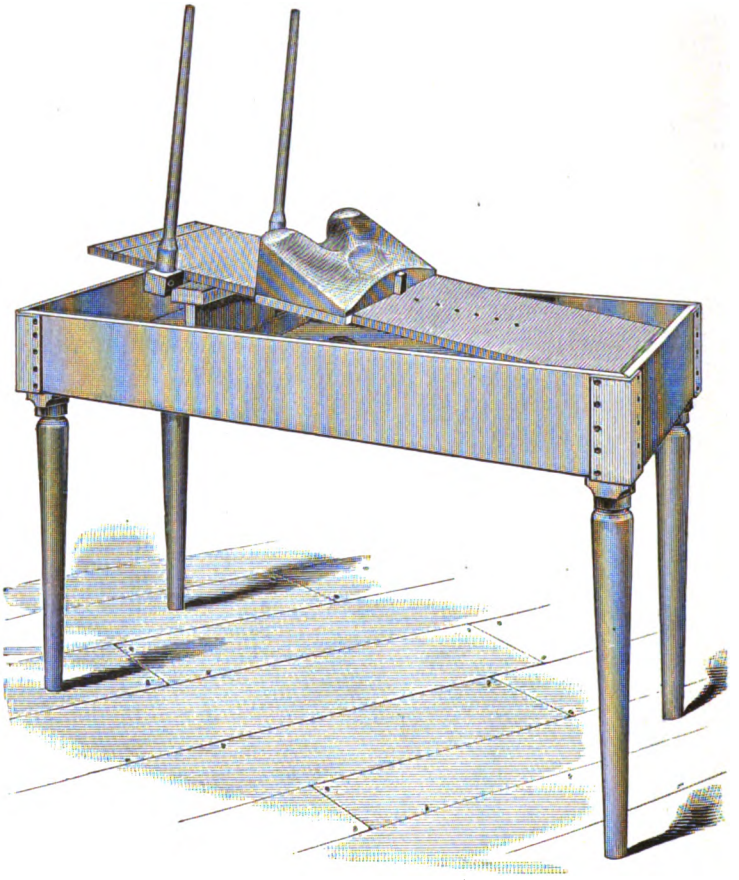
The perineal board (*vid.* Fig. 5), is about 18 cm. shorter than the sink. The height of the patient's buttocks is regulated by the shoulder yoke. By pushing the yoke up the inclined plane the perineum of the patient can be raised to the level of the standing operator's eyes and the patient's back be exposed up to the shoulders.

FIG. 5.



The Operating Table with its Perineal Board.

The yoke (*vid.* Figs. 5 and 6), is made from a solid block of ash, and is carved to receive comfortably the head, neck and shoulders of the patient. The patient's legs are flexed and bear, behind the knees, against the vertical posts.

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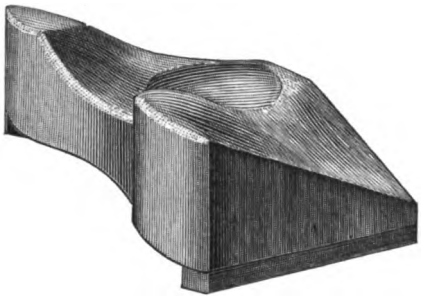
The posts of the perineal board are made of hickory. The table and all of its parts are made of ash and should be thoroughly paraffined six or more times in the first year.

FIG. 6.



The Yoke of the Perineal Board.

THE JOHNS HOPKINS HOSPITAL,
January 20th, 1891.

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DESCRIPTION OF THE PLATES.

PLATE I. The wound as it appears at the first dressing after an operation for the removal of carcinoma of the breast.

The open wound is completely filled with a blood clot.

The little convexities, o, o, and ×, ×, of the skin at the margin of the clot are caused by the approximation sutures which are buried in the clot. The white spots at the upper part of the open wound represent the decolorized parts of the blood clot. The dark spots at and near the inner edge of the open wound represent deeply pigmented areas of the blood clot.

This case illustrates well the use which is made of the triangular flap to cover the defect. The apex of the flap was originally about 2.5 cm. below the centre of the clavicle. It is now almost on the line of the nipples.

PLATE II. The wound as it appeared at the first dressing after an operation for the radical cure of inguinal hernia. The wound extended from + to +.

It was closed with buried skin sutures.

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PLATE 1.

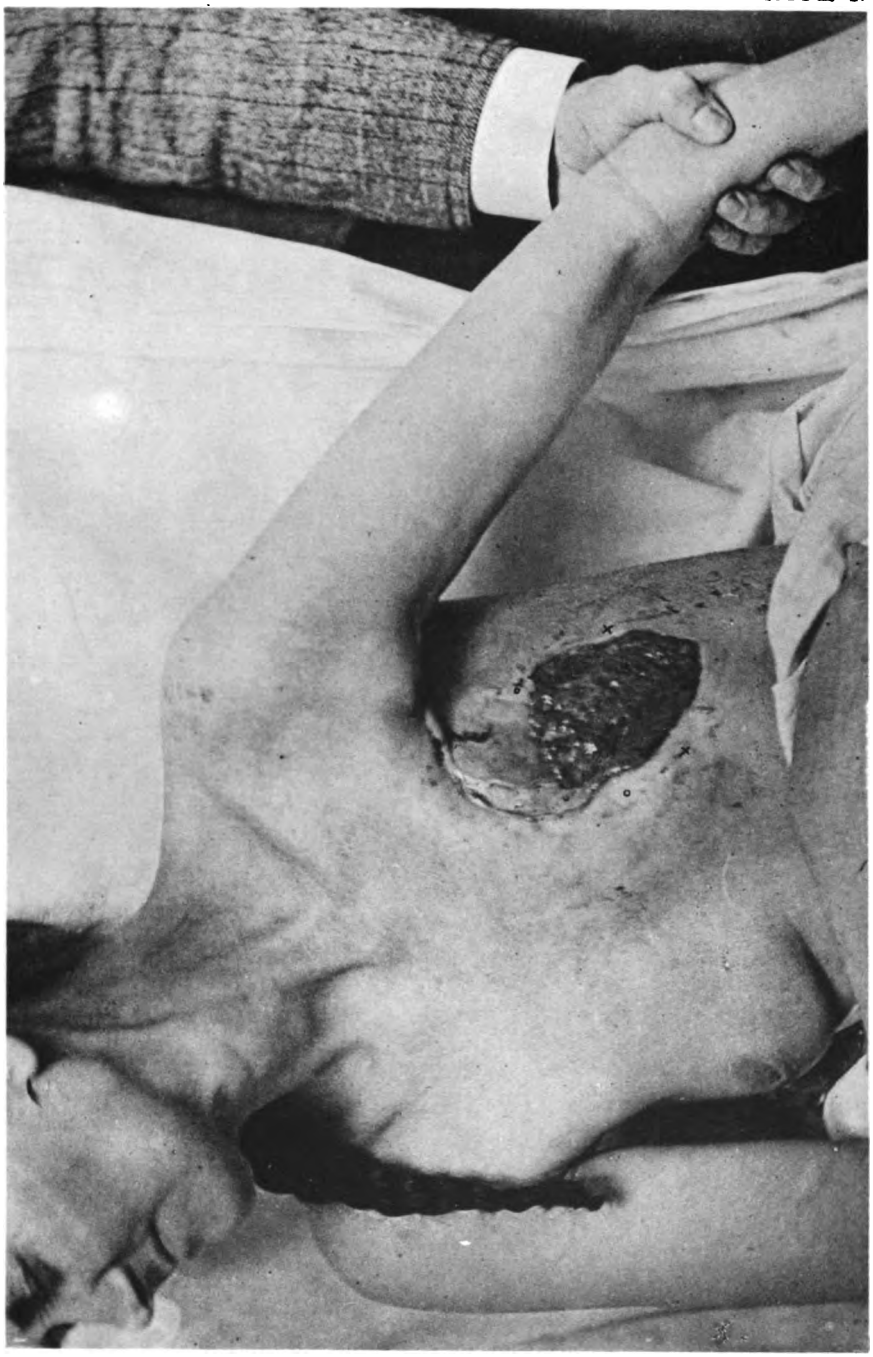

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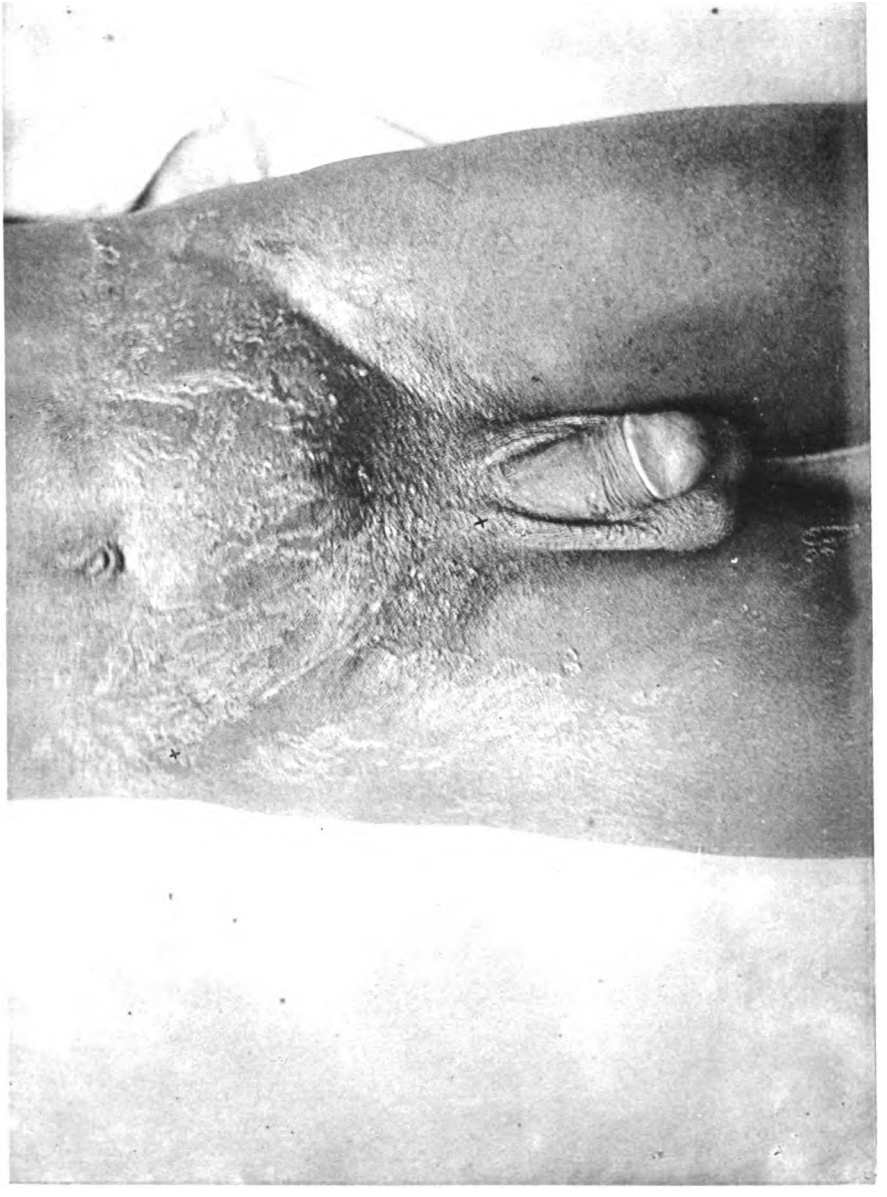
PLATE 2.



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Vol. II No. 6

THE

JOHNS HOPKINS HOSPITAL REPORTS

REPORT IN NEUROLOGY, I

CONTENTS.

I. A Case of Chorea Insaniens, with a Contribution to the Germ Theory of Chorea. By HENRY J. BERKLEY, M. D.
II. Acute Angio-Neurotic Oedema. By CHARLES E. SIMON, M. D.
III. Haematomyelia. By AUG. HOCH, M. D.
IV. A Case of Cerebro-Spinal Syphilis, with an Unusual Lesion in the Spinal Cord. By HENRY M. THOMAS, M. D.

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HOSPITAL REPORTS.

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A CASE OF CHOREA INSANIENS, WITH A CONTRIBUTION TO THE GERM THEORY OF CHOREA.

BY HENRY J. BERKLEY, M. D.

Not many years ago tetanus was considered to be a disease of purely neurotic origin; within the past few months it has clearly been demonstrated to be caused by a specific germ, originating in the earth under certain conditions.[1]

During the past few years there has also been a growing tendency, rather hinted at than openly expressed, to regard the Sydenham chorea as an infectious disease (Strümpell,[2] P. Koch,[3] Eade,[4] Prior[5]); unfortunately, so far without direct evidence, except the finding of cocci in the blood in a single case (Richter[6]) and of rod-like bodies (probably post-mortem products) by Donkin.[7]

Chorea is the common accompaniment of any number of infectious disorders—scarlet fever, measles, whooping cough, typhus abdominalis,

[1] Kitasato. Deutsche Med. Woch., 1889, S. 605.
[2] Strümpell. Specielle Path. u. Therapie, Leipzig, 1883.
[3] P. Koch. Deutsches Arch. f. Klin. Med., XL, H. 5.
[4] Eade. Brit. Med. Jour., 1889, p. 700.
[5] Prior. Berl. Klin. Wochensch., 1886.
[6] Richter. Western Lancet, Vol. XII, p. 529.
[7] Donkin. Brit. Med. Times and Gaz., 1884, II, p. 743.

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varicella, pneumonia, diphtheria, influenza, and many others, making too long a list to follow even to its termination. In the dog it is almost always a sequel of distemper, sometimes coming on as I have witnessed, at the acme of the affection, and rapidly subsiding with the primary disease.

Chorea is commonly supposed to be of non-febrile nature; in the milder forms, undoubtedly, the temperature is not sufficiently high to attract attention, just as in mild rheumatic affections, the rise of temperature is hardly appreciable; yet in Mackenzie's[1] statistics, in 14 per cent. of the cases in which the temperature was noted, there was an increase above the normal of the bodily heat; and in all cases of fatal chorea that I can find, in which the temperature was taken, it varied from 100° to 107° F., averaging about 105°.

Among the complications of chorea that seem to have been overlooked, is the tendency to the formation of abscesses, cellulitis, parotitis, etc., that occasionally manifests itself in this disease, as in other infectious troubles. I cite, *e. g.*, Buzzard,[2] in whose case there was extensive cellulitis of the forearm; Sturges,[3] an abscess on the buttock; Gee,[4] extensive suppuration within the sheaths of several muscles, bilateral parotides, and in a second case also bilateral parotides; all occurring at the height of the disease.

The frequency of endocarditis in St. Vitus dance has given rise to the most common theory of the origin of the disease, namely, the embolic. The more the weight of post-mortem evidence is considered, the less probable does this idea become; cases of chorea are of frequency in which there is no ascertainable—*inter vitam*—sign of endocardial inflammation, and in cases where the so-called embolic dust is scattered widely in the brain vessels, the result produced is not chorea in its proper signification, but convulsive movements and paralyses.

Endocarditis may be in chorea entirely independent of rheumatic origin, having been known to occur in cases ascribed to fright, or it may come on during the course of the chorea and not precede it; an incident accounted for by the very improbable supposition that over-

[1] Mackenzie. Brit. Med. Journal, 1887.
[2] Buzzard. Lancet, 1882, II, p. 847.
[3] Sturges. Med. Times and Gaz., 1880, p. 257.
[4] Gee. St. Bartholomew Hospital Rep., 1886, XXII, p. 89.

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action of the heart causes a rubbing together and inflammation of the edges of the mitral valves; (why not also of the tricuspid?), yet I can find no fatal case in which it has been noted. Would it not be more simple to account for the endocarditis on the basis of a general inflammatory condition arising from an irritant existing temporarily in the blood, for, as will presently be seen, the morbid state of the lining membrane of the heart is not the single change to be found within the limits of the vascular system.

Chorea, like rheumatism, and to a certain extent the exanthemata, has a tendency to return; examples are not infrequent in which there are two, three or more attacks. Finally, uncomplicated chorea may be a mild febrile affection, with a gradual rise and decline of its motor symptoms; accompanied (like diphtheria) by pareses, by mental failure; occasionally by eruptions mainly of an erythematous nature, more rarely with marked symptoms of a pyogenic character; and like rheumatism, is most frequent in moist climates and in the winter months; also like rheumatism, chorea has a maniacal form, the mental symptoms in both affections subsiding during the decadence of the acute symptoms. This peculiar type is now usually known by the name given it by Bernt many years ago—chorea insaniens—of which the present case is an illustration. The opportunity for the examination of the nervous system I owe to the kindness of Professor Welch.

Case I.

History.—Miss A., aet. 27, a teacher by occupation, was admitted to the Johns Hopkins Hospital—service of Prof. Osler—December 24th, 1889, complaining of incoördinate movements of the extremities and body.

Family History.—The patient has three brothers and three sisters living, and in good health. A brother died of pneumonia when young; he had chorea for a month at the age of ten years. Her grand-parents lived to an advanced age. There is no further family history of import.

Previous History.—A. has been generally strong and healthy; had scarlet fever, diphtheria and measles, when a child. Menstruated at the age of sixteen. About eleven years ago she had an attack of

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rheumatism, lasting four weeks; the shoulders and hands were then swollen. She recovered her health completely afterwards. Since that time she has been teaching and studying excessively hard, so much so that at times her friends feared she would lose her reason under the mental strain. A second attack of rheumatism occurred four years ago, after a period of intense application to her studies; during which she became much run down. The hands were much swollen, all other joints slightly so, and became exquisitely tender. She had been ill some days, when owing to some excitement her mind became affected, and for five weeks she was delirious, and was supposed to be suffering from meningitis. During three of these weeks she had the same irregular movements of the legs and arms as in the present illness, though not so violent. These muscular jerkings and mental symptoms disappeared, and she continued in good health until August, 1889, when headaches, which had been troubling her slightly during the previous winter, became more severe. The eyes now often pained her, so that she could not read for any length of time. The back also felt weak after very slight exertion, and she became very nervous. Shortly afterwards she was confined to bed for three weeks, at the end of which time she went away on a visit. During this visit she was able to go about, ride and dance, though still suffering from headache at times. During the night of December 17th, 1889, she was awakened by a jerking of the muscles of the right arm, then of the other arm, finally the legs, face and body were involved. This state has continued unchanged until admission.

Present State.—A. is unable to sleep. The mind is clear. Articulation difficult. She tosses continually from one side of the bed to the other; the movements are very violent. Nutrition is good, as is also the color of the skin. There are extremely well-marked and very varied choreic movements, affecting all parts of the body. On the face and arms, here and there on the legs, sparingly on the trunk, there is a flat papular bronze-colored rash. In places it is distinctly scaly. It is most abundant on the inner side of the wrists, palms of hands and bends of elbows. It is symmetrical. The cervical glands are enlarged, hard and nodular; there is slight enlargement of the inguinal glands on the left side. The pharynx is ulcerated, uvula healthy. Careful examination of the fauces was not possible on

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account of the movements of the head. There is some patchy alopecia. The heart sounds are clear, both at apex and base; the apex beat is in the fifth intercostal space. On the sides and under surface of the clitoris is an elevated and excoriated, non-indurated sore. A further examination of the vagina and cervix was not possible. There is no incontinence of urine or of feces.

December 28. The patient was very restless up to midnight; and then frightened by an imaginary something, jumped out of bed, shrieked wildly and apparently did not recognize the nurse. Was afterwards quiet for a few minutes at a time, but had periods of maniacal excitement, starting at every sound. The muscular movements are not so exaggerated as during the previous night, but the mental condition is decidedly worse. At 3.50 a. m. chloralamide, gr. xxx, was administered, and she became more quiet, and at half-past four was sleeping heavily. The choreic movements cease during deep sleep.

December 29. Patient is somewhat better this morning. The muscular contractions are not so violent. Heart sounds clear, both at apex and base. There is no joint tenderness. Several hypodermics of morphia sulphate were given during the evening; under its influence she slept from nine p. m. until one o'clock a. m. Was then restless until after three, when she slept for three-quarters of an hour.

December 30. The choreic movements are quite general this morning; the left arm, which was quiet yesterday, is now tossed about equally with the right. There is less mental confusion—patient answering questions more rationally. The tongue is moist and furred. Takes nourishment better than yesterday. Under the influence of a third of a grain, morphia sulphate, she slept uninterruptedly through the night.

December 31. Is very restless this morning. The choreic movements are about the same as yesterday. Pupils normal. She speaks very volubly and irregularly; speech is not so distinct as previously. Tongue moderately dry. Pulse 144, not especially weak. Still takes nourishment very well by the mouth. There is a very marked friction erythema on the arms, face, feet and knees.

January 1. Patient slept over six hours last night under the influence of morphia, given at half-past nine o'clock. Pulse 100,

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very irregular. Seemed better this morning; appetite good. Muscular twitchings not so violent. At mid-day she fell out of bed, and when picked up was found to have passed a copious liquid stool. After removal to bed became somewhat cyanosed. Pulse 132, extremely feeble. Ether was given hypodermically, and brandy and hot water by mouth and rectum. At 5.30 p. m. the pulse was still rapid, but of better volume. Is still restless, and has occasional spells of shrieking.

January 3. Since the last note was taken, patient has been in a stuporous condition, lying on back and breathing regularly, 24 to the minute. Pulse 120, not decidedly better than two days ago. There are occasional choreic movements, limited to the arms. Has had several watery stools this forenoon. The abdomen is soft. Tongue and lips are dry and brown. At the angle of the jaw on the right side there is a hard and tender swelling over which the skin is reddened. The temperature has been taken regularly since January 2d, morning, at which time it was 105° in the rectum; at 5 a. m., January 3d, it was $96\frac{4}{10}$°, and at 8 a. m. was $100\frac{4}{10}$°. The heart sounds at apex and base are clear. There are no choreic movements.

January 4. The abdomen is soft and a little distended; there are no spots visible on it. Respiration rapid, 44 to the minute. Fine crepitant râles are present in the left axillary region, with expiratory rub. There is no definite heart murmur. The same fine crepitant râles are present in right axillary region, also with expiratory rub. Patient sank gradually during the day, death occurring at 6.30 p. m.

Autopsy by Dr. Councilman, 14 hours after death.

Exterior Body. Body of medium size, 157 cm. long, slightly built, well nourished. Posterior surface much congested. Right side of face swollen, reddened and oedematous; a considerable amount of fluid blood flowed from the ear. Bloody fluid also escaped from the nostrils. On the anterior surface of the right thigh, and on the internal surface of the left near the knee, are depressed cicatrices. Here and there over the body, generally corresponding to creases in the clothing, are congested areas.

Cranium. Scalp thin and firmly adherent. Skull of ordinary thickness, symmetrical. External surface of dura mater smooth.

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Fluid blood in longitudinal sinus. Dura slightly attached to bone everywhere. The brain with the meninges weighed 1,274 gram. The lateral sinuses are free from clots. The petrous bone of the right side is hyperaemic. Pia mater is slightly oedematous. The arteries at the basis cranii are normal. No abnormal change is to be seen in the cerebral tissues. No marked hyperaemia of the cortex. No hemorrhages.

Abdominal Cavity. The subcutaneous fat is slightly developed. Muscles red. Peritoneum smooth. The cavity dry. The liver and spleen are free from adhesions. Liver, spleen and pancreas congested. The kidneys showed foetal lobulation.

In Douglas' cul-de-sac numerous ecchymoses. The uterus is large, blood vessels prominent, the tissue hyperaemic. Small corpus luteum in left ovary. Immediately around the meatus urinarius, the mucous membrane is slightly swollen. There are no ulcerations or cicatrices about the genitalia.

Thoracic Cavity. Praecordial space uncovered by lung tissue, small. Both layers of the pericardium are smooth. Heart contracted. In the right cavity a small amount of fluid blood, same on left side. Heart flesh pale and flabby. On the auricular surface of the mitral valve are a few warty vegetations. Columnae carnae in left ventricle fibrous. Coronary arteries normal. The lungs are free from adhesions. In the upper part of the left lung is an area of consolidation, the pleura over it being covered with a slight fibrous exudation. On section two foci are seen, the centre of one of them being somewhat whitish. Adjoining them is a depression on the surface of the lung, and beneath this is a caseous focus 3 mm. in diameter, surrounded by indurated lung substance, the induration extending downward into the lung. The lung tissue in the lower lobe is hyperaemic. Pus can be squeezed from all the small bronchi, and there are small scattered foci of consolidation. On the upper border of the lower lobe is a small recent infarction. The surface of the lower lobe of the right lung is hyperaemic. In the upper lobe are numerous areas of consolidation, the largest about 2 cm. in diameter. In the lower lobe pus can be squeezed from all small bronchi. There are also numerous small foci of consolidation. The mucous membrane of the bronchi of both lungs is intensely hyperaemic.

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Parotid Gland. Extensive purulent infiltration of the right parotid. The external ear is intensely reddened and hyperaemic.

Pharynx. The mucous membrane is swollen and intensely hyperaemic.

Spinal Cord. The membranes and medulla are apparently normal.

Microscopic examination of the fresh tissue by Dr. Councilman showed the heart muscle to be slightly degenerated (fatty), the epithelium of the kidneys swollen and granular, fatty in the upper collecting tubes. The liver was normal. Cultures were made from the blood in the right and the left ventricles, and from the valvular vegetations, also from the parotid gland. Those from the right ventricle remained sterile, probably on account of overheating; from the others there was an abundant growth of the staphylococcus p. aureus.

Anatomical Diagnosis. Acute endocarditis of mitral valve. Abscess of parotid. Catarrhal pneumonia of both lungs with bronchitis. Old tuberculosis of the left lung.

The portions of the brain reserved for minute examination, were carefully hardened in Müller's fluid for over three months; and dehydrated in alcohol, both in the dark; and imbedded in celloidin. Over five hundred sections were mounted and examined. All the usual staining agents were employed, carmine, hematoxylin, eosine, Weigert's hematoxylin, acid borax carmine; besides a large list of the analine colors, single, and in combination. Carmine, hematoxylin and eosine, were the most satisfactory for the nervous elements, methyl-violet for the hyaline substance. Macroscopically the brain showed no abnormality on section; in the pons at the level of the exit of the trigeminus were a number of minute hemorrhages among the pyramidal columns.

Microscopic Examination. The convolutions examined were: right, first frontal, posterior portion; paracentral gyri on outer and mesial aspect, convolutions of the island of Reil, ascending frontal middle third, and gyrus fornicatus, middle third; left, gyrus fornicatus, insula, ascending parietal, ascending frontal. There was no essential difference between the motor and sensory regions examined, nor between the right and left halves of the brain, and none between the individual convolutions.

Lepto-meninges. The meshes of the pia are infiltrated in slight degree with round cells, white corpuscles, many of them polynuclear,

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and fresh blood disks, the last being in some places so numerous as almost to constitute hemorrhagic foci. Rather infrequently the walls of the smaller arteries are thickly studded with small round cells. The muscularis is everywhere apparently normal. Here and there in the arteries the intima is in spots thickened, the nuclei swollen, which in the largest vessels distinctly becomes, in places, an acute endarteritis. In the arterioles and veins the endothelial nuclei are simply swollen, and sometimes has separated from the media—a post-mortem change. These endothelial cells, as well as those adherent to the walls, have numerous refractile granules in their substance, which when frequent in the intima of a larger vessel gives a very remarkable appearance to the lining membrane. These granules occur singly in each nucleus, and are most frequently present in the whetstone-nuclei. The vessels while generally containing blood cells are not unusually distended. Their contents vary; besides the separated endothelium, there are unusually large numbers of leucocytes, often arranged in clumps; numbers of them being polynuclear; various coagula, staining blue with hematoxylin and nigrosine; and occasionally in the veins there are long deposits, completely filling the lumen of a perfectly hyaline substance, containing only an occasional white corpuscle; entirely resistant to carmine, fuchsin, the analine blues; occasionally staining faintly with hematoxylin, and only uniformly with methyl-violet. This substance is without reaction to iodine, or iodine and sulphuric acid, and to acids and alkalies singly. The red corpuscles also show a resistance to the staining action of eosin; in some vessels nearly all the disks are colored a rose tint, in in others perhaps a dozen among the hundred in the lumen. The meninges are nowhere adherent to the cortex.

Cortex. Nerve Tissues. The barren layer has in all respects a natural appearance, there being no increase of nuclei, and the fine bands of external fibres present their usual contour.

Second and third layers. The nerve cells are more finely granular than usual, and the cell is often pushed to one side, or end of its space: this pericellular space is large, ofttimes containing as many as three, four or even five amoeboid corpuscles. The processes are well developed, and may be traced for long distances. Rarely there is a vacuole in the protoplasm, still more rarely in the nucleus, both being probably artefacts. Usually the nucleus and nucleolus are present

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with their ordinary characteristics. The fourth and fifth have the same general character as the third layer.

Medullated Fibres. The medullated tubes both in the gray substance, and in the deeper tracts, are brought out clearly in carmine and Weigert preparations, and show no abnormality in contour. The ground substance is not unusually nucleated or granular: careful search reveals comparatively few wandering corpuscles in the tissues, and these mainly in the neighborhood of vessels.

Vessels. There is no noteworthy difference between the vessels of the white and gray substances. The perivascular spaces, while wide, are not unusually so: these spaces, especially at the division of an arteriole, are often filled with accumulations of round cells, occasionally imbedded in hyaline masses. These round cells are pretty uniformly present along the margin of the His' lymph space of all the smaller arterioles. Occasionally there are transudations of red, as well as white blood cells in the lymph sheath, which are rarely sufficient to fill up the space and penetrate into the surrounding tissue. The largest transudations are around veins, not arteries. The adventitia is also often covered by small round cells, and hematoidin débris; leucocytes are seen penetrating the walls and in Virchow's lymph space, which occasionally also contains some globules of hyaline matter. The muscularis is not hypertrophied; the intima has the same thickening, or swelling of the nuclei as has been before noted in the pia, but in less degree; where the endothelial cells are seen flat in the interior of the vessels, the nuclei often have a single vacuole in their centre: this is almost invariable with the whetstone nuclei, in less degree with the oval; perhaps owing to the circumstance that the latter are less frequent on a plane surface. The vessels are only moderately filled with blood, which contains large numbers of white corpuscles, and has the same eosin reaction as before mentioned. A few of the leucocytes have a distinctly hyaline deposit in the centre. The veins contain more blood than the arterioles. The round cell proliferation is seldom seen upon the walls of veins, but, on the other hand, the extravasations, hyaline deposits in the lumen, and globules of it in the lymph space; as well as a peculiar detritus in the same, are more marked. The meshes of the trabeculae connecting a vessel with the neuroglia, are sometimes filled in arterioles with the hyaline substance, arranged in globules; or at times coalesced into masses,

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divided by thin irregular lines. The fine connecting capillaries are generally filled with a single row of red blood disks, with occasional large leucocytes apparently plugging the whole calibre, and more rarely droplets of hyaline material. There is no increase of nuclei in the capillary wall.

Optic Thalami. Sections taken from different portions of these bodies show normal ganglion cells, with a few leucocytes in the lymph room, though not so many as in the cortex. The nerve fibres passing in all directions have their usual number and contour.

The vascular state is unchanged, except that the round cells and hematoidin débris are somewhat less frequently met with; but when present occupy larger areas of the perivascular space.

Internal Capsules. (Middle third). There is no visible alteration of the medullated fibres.

Lenticular Ganglia. The same condition is present as in the thalami.

External Capsule and Claustrum. Nerve elements are normal.

Corpora Quadrigemina (anterior). The general condition of the nerve tissues and vascular system corresponds with the central ganglia.

Optic Commissure. The fibres of the optic nerves are normal, the connective tissue nuclei among them are rather prominent. In the surrounding meninges there is an extensive infiltration in foci of round cells; sometimes the vessels are so thickly studded with them, as only to be distinguished by their course. Around the vessels, especially veins, there are extensive extravasations of red and white corpuscles, and at times quantities of blood débris and fine granular detritus. In places along the nerve sheath, blood cells are from five to six rows deep. Other characteristics are the same as on the cortex.

Cerebellum. The only portion of this organ examined was a small piece that had been left attached to the medulla. Excepting the slight vascular changes it appeared to be quite healthy.

Pons Varolii (level of exit of trigeminus). Along the convexity of the pons, the pia is strewn quite thickly with red blood corpuscles; especially is this the case near the exit of the fifth pair, where they are grouped in masses, together with heavy deposits of small cells. This cell infiltration is mainly in foci, but the meninges are not adherent to the medullary substance. The nerve fibres of the tri-

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geminus have retained their natural appearance, though the nuclei among the root fibres are perhaps a little increased in numbers. The endarteritis in places is a very striking feature, especially in the basilar artery. Even in this vessel the extent varies considerably in different sections: where it is least developed the thickening is confined chiefly to the pons side of the artery, in others it spreads over the whole of the interior of the vessel, extending into branches given off in its course. The adventitial change is slight in comparison with the internal, consisting solely in a slight penetration of the layer with round cells.

Within the substance of the pons the same vascular condition is continued, modified as in the cerebrum. The hyaline masses around arteries and veins, filling up of perivascular spaces (especially of veins), with red and white corpuscles, and spheres of hyaline material within the lumen, reach their acme in this region. Occasionally one is almost reminded of a thrombotic process by the peculiar arrangement of the hyaline in the veins. For example, a vessel contained three hyaline spheroids, filling the lumen; centrally was a large mass of leucocytes, with very few red disks, while beyond the hyaline the red cells contained very few white corpuscles mingled with them.

A new feature now appears in the form of numerous foci of minute hemorrhage, distributed through the central and anterior portions of the pons, but especially located among the fibres of the pyramidal tracts. In these hemorrhages the blood is often quite fresh; the blood cells while distorted are not disintegrated, nor are the amoeboid cells over frequent: others contain centres staining more deeply with hematoxylin than the periphery, and hold many small, round nuclei, among tissues seemingly beginning to break down. In Weigert's preparations this centre remains unstained. From ten to seventeen hemorrhages were counted in each section mounted, mostly microscopic, the largest not being equal in size to a mustard seed.

The nerve and neuroglia tissues are everywhere natural, except for the infiltration with red blood cells in the neighborhood of the hemorrhages.

Medulla Oblongata. (Sections were made at various levels from the highest part of the hypoglossal nucleus to the decussation of the pyramids.) The meninges gradually show, as the sections progress

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downward, fewer foci of infiltration and hemorrhage; until near the decussation, both are lost. In several of the larger arteries there is still distinct endarteritis. So far as the pia is concerned, there is a difference between the higher and lower regions of the bulb. The hyaline matter and blood cells are still present around some of the vessels in the interior of the medulla, but there are no infiltrating hemorrhages in the tissues. The nuclei on the floor of the ventricle, and more anteriorly, the olivary bodies, the internal fibres of the vagus and hypoglossus, as well as the transverse fibres, were examined with great care and patience, but without finding the slightest pathological change in them. In the pyramids, especially in their middle and outer portions, apparently not more numerous on one side than the other, are a few degenerated nerve fibres, apparent both in Weigert and carmine preparations. With carmine the substance of Schwann is colored rather deeply by the dye, with the axis cylinder much less distinct than its neighbors; or the cylinder may take the same tint as the medullary substance, so as not easily to be distinguished from it. There is no increase of neuroglia.

Spinal Cord (cervical region). The medulla spinalis is throughout its length of typical form, both in the white and gray tracts. The pia over the whole area of the cervical enlargement is healthy, only, there is around the central artery and vein a slight transudation. Sections were taken from the highest cervical, middle of cervical enlargement, and from the origin of the seventh nerve. The nerve roots are normal, axis cylinder, and myeline perfect. The post-mortem separation of the endothelial lining is not so marked as in the cerebral lepto-meninges. The arteries of white and gray substances generally contain little blood; the veins are moderately injected; neither seem to have a morbid change in their sheaths. Frequent hemorrhages in the gray columns of both sides are met with in the mid-cervical region, extending over a length of nearly three centimetres. These are always around thin walled veins, and isolated fresh blood cells have penetrated a considerable distance from the vessel. In the white columns there are none. The hyaline substance has disappeared from around the vessels, a few small veins were found plugged with refractile masses arranged with peculiar concentric laminae.

In the pyramidal tracts the scattered fibres, degenerated singly in the pyramids, have now joined in groups of three or four. In each

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indirect column there are from three to four of these spots, always in the posterior and central portion; in the left direct fascia there is a single one, almost on the margin of the anterior commissure. In these spots the myeline tinges slightly with carmine, the axis cylinder is indistinct, the whole fibre seems shrunken, while between the individual fibres the neuroglia is a little thicker and more granular than elsewhere. Not a single altered fibre could be found in any of the other columns. The multipolar cells are mainly perfect in protoplasm, nucleus and prolongations; some eight or ten cells, very granular, without nuclei or protoplasm, shrunken to a small mass, were found in the total number of sections examined of this region. The neuroglia has no increase of nuclei, the axis cylinders in the gray horns are in their usual numbers and stain readily.

Dorsal and Lumbar Cord. The general appearance is the same as in higher regions; even fewer granular cells are to be found. There are two minute hemorrhages in the left column of Burdach, in the upper dorsal segment; and two in the right column of Goll in the superior lumbar segment; none in the gray horns. The meninges rarely have inconsiderable foci of round cells, but there were no vascular lesions discovered. The central canal is patulous through its entire length. In the lower cervical region there is a slight erosion of the epithelium lining the canal, on the ventral side. The areas of degeneration disappear in the highest lumbar segment.

Analysis.—Paramount among the changes that may undoubtedly be considered as morbid is the meningo-vascular. The focal inflammation, between the gyri, at the base, and around the pons, and in less degree the patches on the cord, are significant of an infectious trouble; nor are the infiltration of these same spots with dense deposits of small round cells around the vessels, the swelling of the meningeal and endothelial nuclei; the infiltration of the membrane with red, uni- and polynuclear leucocytes; and more rarely a corpuscle closely allied to the granular, less so. Significant, too, is the peculiar endo-arterial inflammation in so young a subject, patchy even in the basilar artery; affecting some of its branches for short distances, others not at all, apparent in a few of the arteries of the cephalic-meninges, absent from the vast majority; represented in the arterioles solely by a more or less pronounced swelling of the endothelium, disappearing entirely in the capillaries. Such a condition could only be produced by the

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action of some pathogenic germ, or the soluble products of the same.

The contents of the vessels, and perivascular spaces afford a slight but rather indicative link to the chain; the red blood corpuscles showed no positive alteration, the eosin reaction being very indefinite, possibly depending on some peculiarity of the hardening media; more positive are the numbers of leucocytes, accumulated at times to somewhat dense aggregations within the vessel, and penetrating the wall in numbers. Blood coagulations assume so many forms that it is difficult to make any deduction from them (I do not refer to the hyaline material), but besides, the rarer detritus in the vessels and more common in the spaces, remind one of the old deposits once of unknown nature, now generally conceded to be accumulations of organisms.

The red-cell extravasations I attach no especial importance to, they being commonly found like pigment deposits in the brains of persons dying of a variety of diseases; and I have seen the same even more pronounced than in this case in the cerebrum of a person who died of typhoid fever.

The agglomerations of round cells within the margin of the perivascular lymph spaces, especially at the branching of arterioles, are, I think, of some import. These nuclei vary greatly in every brain, sometimes fewer, sometimes greater in numbers, but in this instance they exceed by far the average, and constitute an object striking to the eye in the histology of the vascular system.

The significance of the refractile granules in the centres of the endothelial nuclei is unknown to me; in every brain they may be seen in small numbers if careful search be made for them, but they are not prominent as in this chorea brain, looking in every vessel as if the nuclei had holes cleanly punched through them, or that vacuoles were present where a former nucleolus had been.

In the nervous and connective tissues of the cerebrum, alterations are so slight as almost to be passed without comment; the nerve fibres were apparently absolutely normal, the cerebral cells were certainly a little more granular than ordinary, but unfortunately we are not certain if this be a result of overaction or no; the lymph spaces around them were larger than normal, and contained more wandering cells than is usual, even in typhoid fever; all bespeaking a slightly oedematous condition of the cortex, lessened in the basal ganglia.

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Throughout the cerebro-spinal axis small hemorrhages were found, consisting (with few exceptions and these solely in the pons) of quite fresh red corpuscles infiltrated into the tissue. Now these small hemorrhages are entirely without significance, almost every cerebrum and cord contain some of them, and they have no reference to any single disease; being found indifferently, for example, in tetanus and pseudo-hypertrophic paralysis, only in spasmodic affections they occur in greater numbers than in non-spasmodic, a difference probably due to the muscular contractions driving the blood from the extremities to the viscera. They probably occur at the moment of death, and even in a healthy dog that was chloroformed and bled to death from the jugular vein, a few were found in the gray horns of the spinal cord.

Somewhat different were the few older hemorrhages in the pyramidal regions of the pons, with their nuclear increase, unstained centres and altered blood cells, a result that must have taken several days to accomplish, especially to cause the degeneration of the pyramidal fibres in the medulla and cord, undoubtedly their direct result.

Müller's fluid preparations are by no means the best to look for organisms in, but in the region of these ruptures every care was taken and method used to ascertain whether there remained any bacteria in the neighborhood of the hemorrhages or in the meninges, but without success, except for the uncertain discovery of a few scattered cocci in a single hemorrhagic focus.

The last and most universally pervading alteration is the hyaline. In the microscopic description I have not mentioned a peculiar round or oval, chalky-looking body, which, with high powers, has somewhat of a laminated arrangement, existing only in the perivascular spaces. These bodies, though resistant to acids and alkalies, stain with hematoxylin a uniform blue, and with Weigert's hematoxylin assume the same reaction as the myeline substance of the nerve tubes. They not only are present in chorea and typhoid fever, but also in perfectly normal brains, and are doubtless produced by some unknown quality of hardening agents, being found equally in alcohol, Müller, and Flemming preparations, though most numerous in alcohol ones, probably being brought out in the others by the after alcohol dehydration, in the same way as the lethecine balls, and to a less extent many bodies that are commonly designated amyloid,

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though these especial forms bear but a small resemblance optically to the latter, much more resembling the myeline drops in acute softening when stained with Weigert's method, though often much larger.

Quite different in reaction is the perfectly homogeneous drops and concretions, both within and without the lumen of vessels. It is not a common find in examinations of the cerebrum, having been seen most frequently in progressive paralysis, but when the cerebrum is excluded in many infectious diseases. More closely allied to glycogen,[1] both histologically and in some of its chemical reactions (resistance to acids, alkalies, etc.), it is yet wanting in the peculiar reaction of that substance to iodine. Of the genesis of the substance, despite the numerous researches, nothing positive is known; von Recklinghausen,[2] Holschewnikoff[3] and others see in it the result of a metamorphosis of cellular elements, sometimes of adventitia, media, or intima. Oeller[4] ascribes it to red blood corpuscles that have assumed a homogeneous character, not only those in the lumen, but also in the perivascular space, where they have entered through diapedesis. A few writers regard it as a post-mortem product (Wedl,[5] Adler[6]). No one attempts to give a solution of the cause of the hyaline swelling of the cellular elements, nor why the red blood cells become homogeneous. Post-mortem product in the strict sense of the word, it certainly is not; being at once too common and too rare, frequent in the liver for example, uncommon in the brain. Our present case contains argument for both the remaining theories; for the metamorphosis of the cellular elements in the swelling of the endothelium, multiplication of elements in the adventitia and diapedesis of the blood corpuscles through the vessels' walls, but the actual alteration of the endothelial or other cells is wanting; on the other hand, for the blood, there is a certain transparency and non-reception of the eosin stain, a perfect absence of the red cells in the concretions, both in the lumen and spaces, and quantities of them in places where there is little or none of the hyaline globules and masses.

[1] Langhans. Virchow's Arch., Bd. 120, H. 1, 1890.
[2] Von Recklinghausen. Handbuch der Allg. Path., 1883.
[3] Holschewnikoff. Virchow's Arch., Bd. 112, S. 532.
[4] Oeller. Ibid., Bd. 86, S. 329.
[5] Wedl. Cited by Holschewnikoff.
[6] Adler. Arch. of Psych., Bd. v.

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The hyaline droplets in the capillaries also look as if they were derived from the red cells.

Aside from strictly post-mortem change the question of the artificial production of this substance must arise. We are ignorant of what combinations may occur between the common hardening agents, Müller's fluid and alcohol, with the fluids existing in the brain after death. What acids, alkalies or combinations of the same may be there, we do not know, and what may be the result of their combination with the hardening media, we also do not know. Certain it is, however, that under peculiar circumstances a substance closely resembling the hyaline may be formed by certain hardening fluids out of the red blood corpuscles. Portions of a brain hardened in alcohol and Müller's fluid showed no hyaline masses, while in another portion of the same cortex, hardened in Flemming, there were numbers of slightly refractile transparent bodies, both in the lumen and perivascular space (which elsewhere contained slight red-cell extravasations). These bodies were mainly round and separate from each other, but some had coalesced into larger droplets, and all stages could be traced from a red corpuscle to a fully developed transparent globule, five or six times the size of a red disk. In a single vessel the whole calibre was actually filled by the coalesced transparent droplets, and even held a few leucocytes in the mass. As neither osmic, chromic nor acetic acids singly will produce the same result, it must be referred to the combination of all acting in a manner separate and distinct from any single one.

As the uniformity regarding the hyaline formation is by no means great, it is best to consider it for the present—when there is no actual nyaline change in the arterioles' walls—as being of little value, and rather the result of a peculiar chemical change, possibly in some instances induced by the soluble product of pathogenic germs, than to actual disease formation strictly speaking.

Case II.

The chances of the laboratory at this time offered a dog with a well-marked but somewhat old case of chorea. If the germ theory of the causation of the disease be correct, it could afford but slight

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indications (after the lapse of so many months) of an acute process, but as fatal chorea is rare, every step, however obscure, may be of some little importance; it was determined to see what evidence his nervous centres afforded.

"Rake," a full grown male English setter, has had general chorea for between five and six months. The origin of the trouble is unknown. In motion or even standing the jactitations are somewhat rythmical; when reposing they are decidedly arythmical. In the last few weeks of his life he was negligent and dirty in his habits. Appetite always good. Slight general emaciation, with some loss of muscular power.

Chloroformed May 8, 1890. Respiration ceased before the heart's action. Both lungs and pleurae normal; slight thickening of one cusp each of mitral and tricuspid valve, most marked in the tricuspid, where the gelatanoid mass extended downward on the chordae tendinae. Heart muscle healthy. Abdomen: Fat moderately developed, peritoneum smooth, liver, spleen and pancreas healthy. Kidneys studded with small white spots, mainly cortical, underlying the capsule, others deeper. The small intestines contained a few parasites (tricocephalus dispar). Bladder normal, contained a few cubic centimetres of urine which showed no albumen with heat and acid. Brain and spinal cord also the peripheral nerves apparently healthy. Leptomeninges not very transparent.

Agar agar tubes were inoculated from the kidneys, spleen, gall, bladder, and heart blood by Prof. Welch. After five days the bile tubes were the only ones that did not remain sterile, but on the contrary showed well-marked growths—diagnosed—colon bacillus.

Portions of the brain and cord were hardened in alcohol, Flemming, and Müller's fluid; portions of the thoracic and abdominal organs in alcohol and Müller.

Sections of the brain were made from the anterior, middle, and posterior portions, also from the cerebellum. The nervous and neuroglia tissues were normal, nerve cells and processes well developed. The perivascular spaces are wide with occasional extravasations of fresh red blood cells in the sheath, where there is also a considerable number of hematoidin clumps. The coats of the vessels have no

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noteworthy departure from the normal. The pia, more particularly over the anterior and middle portions of the hemispheres, is somewhat thickened. The fibres of the membrane are swollen with large lymph spaces between them (oedema), the nuclei are ofttimes swollen, the meshes are everywhere strewn with red blood cells, some fresh, some old and shrunken; leucocytes abound, many large and very granular, resembling Gluges corpuscles; others contain pigmented bodies, which in the Flemming sections are stained black, and with high powers are very striking objects. There are also very numerous round and irregular bodies, from the size of a red blood cell to four or five times that diameter, granular-looking in unstained Müller's fluid, black in Flemming hardened sections, thickly strewn over the pia. These fatty bodies seem to be normal in the pia of the dog, but not in the same numbers as in the present example. The vessels so far as can be positively determined are without alterations, except here and there a slight uncertain thickening of the intima.

Cord and Bulb. The nerve and neuroglia elements as well as the vessels of the cord are sound. There are occasional extravasations around thin walled veins. The pia is slightly infiltrated with lymphoid cells, but not so much as in the brain. Two rather large vessels running over the pons are very thickly covered by round cells. The central canal is patulous.

The liver, spleen, and gluteal muscles, as well as the sciatic nerve show no signs of any degenerative process. The thickening of the tricuspid was a simple chronic oedematous condition very common in dogs. The structure of the kidneys, except the whitish spots, was perfectly normal. These spots averaged one, by a half millimetre; and are formed of small round cells, with a good many newly-formed blood-vessels. A few shrunken glomeruli, are occasionally found inclosed by the round cells. The blood-vessels everywhere contain large numbers of white cells.

In this second case but two alterations of any value were found, of which the pial is the most important; the white corpuscles bearing pigmented granules; the large granular leucocytes; the oedematous separation of the fibres of the membrane; the solitary *foyer* on the pons, containing the two infiltrated vessels, are tolerably certain

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evidences of a recent inflammation of the pia, now subsiding, in which the leucocytes are enclosing and bearing away the débris of the struggle between the tissues and the microscopic organisms. The hematoidin masses in the vascular lymph sheaths, also show that there was a precedent congestion. The round cell foci in the kidneys may also have been caused by aggregations of organisms, which, after dying, left the shrunken glomeruli and caused the growth of the granular tissue. The cells of the spinal cord did not support the theory of Dr. H. C. Wood,[1] being in all respects perfectly normal. Professor Welch, to whom I submitted the sections of cortex and kidney, was strongly of the opinion that the damage had been caused by germ infection.

A glance over the journal literature of chorea for the past ten years reveals but little uniformity in the post-mortem accounts; congestion of the meninges and brain, as well as other viscera, is most common, even this is sometimes absent; cardiac vegetations are the most usual find, but do not uniformly depend on coëxistent rheumatic trouble, as in Ross'[2] case, nor is it necessary that the choreic movements should be of long duration, for in Beale and Cooke's[3] observation, in which death supervened after the movements had lasted only 130 hours, patches of punctiform congestion and lymphy vegetations were found on the heart valves.

The meningeal condition, both macroscopically and microscopically, in chorea seems, from the literature, to have been almost universally neglected, often not even mentioned; occasionally hyperaemia, oedema, and hemorrhages in it are met with, Naunyn[4] found fungi on the pia; Dana[5] simply mentions that there was some lepto-meningitis, but these are exceptional.

Finally, I will set side by side for comparison the microscopic changes in the present cases with those of diphtheria, taking the description of Babes,[6] the most recent writer on the subject.

[1] Wood. Trans. Coll. Phys., Phila., 1886, VIII, p. 157.
[2] Ross. Canada Med. and Surg. Journal, 1882, XI, p. 197.
[3] Beale and Cooke. Brit. Med. Jour., 1888, p. 795.
[4] Naunyn. Mitth. a. d. Med. Klin. zu. Königsburg, 1888, S. 296.
[5] Dana. Brain, 1890, April.
[6] Babes. Virchow's Arch., Bd. 120, 1890.

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Diphtheria (*Liver and Kidneys*).	*Chorea* (*Meninges, Brain and Kidneys*).
General hyperaemia of internal organs.	Idem.
Focal increase of round cells in the neighborhood of small vessels.	Distinct patchy endo-arteritis: focal increase of round cells in the neighborhood of small vessels.
Collections of leucocytes in the lumen of vessels.	General increase of the number of leucocytes in vessels.
Swelling of the endothelium.	Swelling of the endothelium.
Cloudy swelling, and focal degeneration of the parenchyma cells.	Slight oedema of meninges and brain, granular and fatty degeneration of the kidney's epithelium, fatty degeneration of the heart-muscle.
Beginning hyaline degeneration of the same.	Hyaline globules in the lumen of many vessels, hyaline masses around the same, granular detritus.
Hyaline degeneration of vessels, hyaline globules in lumen, granular detritus.	Leucocytes in meninges inclosing fatty particles. Round cell foci in the kidney (dog).

In conclusion it would seem that chorea might be considered as a general systemic affection, acting with greatest intensity on the vascular system and lepto-meninges, and that its cause is to be sought for in an especial bacillus or its toxical product, not a coccus; the staphylococcus aureus being only an accompaniment to the other germ, as in many other infectious diseases.

The so-called "choreakörperchen" of Flechsig[1] and Wollenberg[2] are, so far as I am able to judge from the first writer's description, by no means peculiar to chorea; if I mistake not, they are to be found not only in the globus pallidus, but in other parts of the brain, not only in this affection, but also in normal as well as diseased ones, and are most probably artefacts.

BALTIMORE, July, 1890.

[1] Flechsig. Verhandlung d. Cong. für in. Med. Wiesbaden, 1888, II.
[2] Wollenberg. Berl. Gesell. f. Psych. u. Nervenk., Mai, 1890.

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ACUTE ANGIO-NEUROTIC OEDEMA.

By CHARLES E. SIMON, M. D.

It is only within the last ten years that the disease, variously described as angio-neurotic oedema, acute local, and acute circumscribed oedema, has begun to receive the attention and study which it deserves, and, in fact, it may be said that it has been recognized as a distinct affection for the first time by Quinke in 1882, who called it 'Acut umschriebenes Hautödem.'

Cases had been reported in 1879 by Milton, under the name of Giant Urticaria, and in 1882 Dinkelaker in his inaugural dissertation 'Über acutes Hautödem,' gave a careful compilation of the cases reported up to that time. Since then reports have been made by various observers, as Jamieson in 1883, Graham and Strübing in 1885, Falcone in 1886, Riehl and Metas in 1887, Osler and Elliot in 1888, and more recently Hartzell; but these references practically cover the literature on the subject.

The disease is characterized by the occurrence of rather circumscribed swellings, coming on with great rapidity, usually multiple, attacking by preference the eyelids, lips, hands, feet, genitals and buttocks of the patient.

As a rule there are no premonitory symptoms, but general lassitude, headaches, impairment of the appetite, may precede an attack; sensations variously described as itching, burning, prickling, etc., are usually experienced, before the swelling appears in a part, which gives place to a general feeling of distension, accompanied in some instances by a certain amount of pain, while in others there is merely a sense of stiffness, when the swelling is at its height. Red spots and streaks occasionally take the place of these swellings, and may precede them, but this can hardly be said to be the rule.

The swelling itself comes on with great rapidity, reaching its maximum in about an hour, or sometimes less—in one of my own cases in ten minutes. At its height the parts are intensely swollen, so

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much so that the eyes may become closed completely when the disease, as it often does, attacks the lids; in the hands movements of the fingers may become altogether impossible.

The color of the swollen parts may be of a slightly reddish hue, but more often it is somewhat paler than normal, as a result of the pressure of the transuded fluid upon the smaller blood-vessels; the temperature is at the same time slightly lowered, following a preliminary rise in the affected parts.

This state of affairs may persist for 1–4 days, continuing uninterruptedly through days and nights, but at times the swelling disappears during the night either entirely, or to some extent, to recur the next morning.

While attacking epidermoid structures, as a rule, it is important to note that the mucous membrane of the pharynx and larynx may also become involved, and while the transudation in the aforementioned structures occurs with such rapidity that it very soon causes the disappearance of any preceding hyperaemia from observation, it is in the fauces and pharynx that we find evidence of a coincident hyperaemia. Again, while the disease in its ordinary form is connected with no danger, as far as life is concerned, it assumes a most serious aspect when these parts become affected. Fortunately such cases are rare, and while no doubt the mucous membrane in these situations is involved to some extent in every case, as is shown by increased redness and slight swelling, it is rather uncommon to meet with cases where it goes on to such a degree that breathing becomes difficult, and even more so that death becomes imminent, or actually occurs. Still, there are several cases on record in which scarification became necessary, and Osler in his series of cases reports one death from sudden oedema of the glottis, due to this cause—case 2 of the 4th generation.

Swallowing is also very rarely interfered with; still, Riehl reports a case of this nature, associated with considerable dyspnœa.

In addition to these features we very frequently observe the occurrence of nausea, vomiting, colicky pains and constipation during an attack, and diarrhœa towards its end, and these symptoms are more frequently present than absent, and attacks of this nature may even take the place, so to speak, of the swellings, or rather occur by

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themselves, thus closely simulating the gastric crises, described by Leyden, which latter again closely resemble those described by Charcot as occurring in some cases of tabes, a case of this nature being reported by Strübing—No. 2—in which the oedema was of rather rare occurrence, while the boy, aged 16, at the time of observation was the subject of attacks of vomiting, occurring quite regularly from his 3d year on.

The vomiting may be slight, but is at times extremely profuse, and after the contents of the stomach have been brought up, bile-colored, watery ejections occur in great quantities.

The pains are, as a rule, proportionate to the severity of the attack; the abdomen is usually drawn in, and the epigastric region, especially at the beginning, rather prominent.

These features are, however, not constant, and in both of my cases absent.

With the approaching end of the attack all the symptoms disappear; a slight degree of lassitude and anorexia may persist for some little time, after which the patient is quite comfortable again, and remains so until the next attack. The swelling itself gradually disappears and usually in the parts first affected.

Particularly interesting is the apparent hereditary tendency of the disease, shown beautifully in the series reported by Osler; he was able to trace the disease through five successive generations, the percentage of members affected in each generation being 50 per cent. in the 2d, 41.1 in the 3d, 90 per cent. in the 4th, and 75 per cent. in the 5th. This same hereditary tendency is also shown in case I of Quinke's, case II of Strübing's and in Falcone's case.

The disease usually begins at an early age, and seems just as common in the male as in the female.

Attacks of this nature occur as a rule with considerable regularity, thus in Metas' case an attack came on every day at almost the same hour; in others they occurred every 7th, 12th and 14th days; in one of my own cases every month, while in others again they come on at irregular intervals, and one attack, as it were, predisposes to another, so that the disease continues indefinitely and has a special predilection in its outbreak for parts previously attacked.

In connection with causes bringing on an attack it is interesting to remember the curious circumstances under which angio-neurotic

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seizures appear. Referring to angio-neuroses in general I may cite two curious cases, in one of which the smoking of a moderately fresh cigar is followed by a most intense blush of the face immediately about the eyes, taking in the upper half of the nose and persisting for half an hour. In the other the ingestion of fish invariably produces oedema of the pharynx accompanied by a sense of suffocation. One of Osler's cases could not eat apples without bringing on an attack of oedema. In other cases indigestion, fatiguing bodily exercise, colds, psychical excitations, sudden barometric changes, etc., seem to stand in a causative relation to the disease.

To pass on now to the cases observed at the Medical Clinic :—

Case I.—Katie S., aet. 15, of German descent, applied at the neurological clinic for treatment, complaining of a marked swelling, affecting her entire left hand and forearm. Nothing like this trouble had occurred in her family, as far as she and her mother could tell. Her father died of phthisis some years ago. One sister has been the subject of chorea three times. The patient had the usual diseases of childhood, also intermittent fever at the age of 5, followed shortly after by pneumonia. Three weeks after recovering from this she had her first attack of swelling, and has been afflicted ever since. The parts affected in her case are the hands, the forearms up to the elbow joint, the feet, the legs up to the knee, and the face. Other parts are never affected, and mere localized swellings never occur. The disease in fact seems quite fanciful in her case, for whenever one of her feet is attacked—and both feet are never attacked together—then the hand and forearm on the opposite side of the body are invariably implicated, both mother and child being quite positive in their statements as to this point. One foot alone, or both feet, or both hands are never attacked at the same time. On the other hand one hand with its corresponding forearm is quite frequently attacked without involving the opposite foot.

When the swelling attacks her face it starts in the eyelids and gradually involves the entire face, travelling from the eyes downward toward the chin, the forehead being the last portion affected. At times the swelling has been so intense as to completely close her eyes, so that she had to be *led* home from school.

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Such attacks have occured in her case with tolerable regularity every month, and the mother affirms that the girl will invariably have two attacks in a month if she has gone without one the month preceding.

In this way the disease had continued until about 20 months ago, when she had a period of rest lasting 1½ years; on April 7th of this year the trouble returned, and at the beginning of the next attack, which occurred on the morning of the 28th, she applied for treatment.

Premonitory symptoms occurred in her case but once, and that before the attack on April 7th when she complained of general lassitude, pains in the back and head for 24 hours preceding. The onset of an attack she can predict ten minutes before the swelling appears by a prickling, itching and slightly burning sensation in the part to become affected. The swelling then appears and progresses with great rapidity, reaching its maximum in about fifteen minutes; while this goes on she experiences a sensation as though something were moving up and down in the part; as the maximum is reached there is slight pain and a feeling of distension, which continues throughout. The part is tender to the touch, and movements at the elbow and knee-joints are somewhat interfered with, active movements causing considerable pain.

When the hand is affected the swelling is diffuse, most marked on the dorsal surface; the fingers are kept in a slight degree of flexion and are intensely swollen. Active movements are impossible, and passive movements cause a great deal of pain.

These swellings last from 1 to 4 days, often disappearing during the night to return the next morning, but not invariably so, and not always completely. As a rule it begins to disappear in the portions first affected, the process being accompanied by an itching sensation.

In her case I was unable to get any history of shortness of breath, or nausea, vomiting, etc., the girl feeling perfectly well throughout the attack.

Her condition on application to the dispensary was the following:

Patient is a well-nourished, intelligent girl, but a little backward in bodily development, considering her age; she has never menstruated. Her eyes are clear, and her general behavior straightforward, and by no means nervous. No abnormalities were noticed about the head.

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Her lungs and heart are perfectly normal.

Her left hand was very much swollen, the fingers flexed to some extent, the color of the skin a little paler than normal and the temperature of the part appeared slightly raised. Between the two wrists there was a difference of 2 cm., and between the hand immediately above the metacarpo-phalangeal joints of 3.1 cm.; the forearm was implicated to a slight extent. The fauces and posterior wall of the pharynx appeared reddened and slightly swollen; there were small vesicles on each side of the uvula, on the velum palati.

On April 29th the girl stated that the swelling had continued throughout the night; the forearm was very much swollen, and the hand slightly more than on the 28th. In the morning her arm hurt her a great deal, and there was a burning sensation in the parts all day.

During the night from the 29th to the 30th the swelling had gone down in the arm, but not in the hand, and the parts pitted on pressure for the first time.

During the night of the 30th she slept badly on account of itching of the face which made her think the swelling would start there also. In the afternoon the swelling had gone down very much in the hand, enabling her to straighten her fingers to almost a normal extent; a little oedema, however, still remained on the dorsum. The forearm had nearly returned to its normal size. The face appeared a little puffed and hot, and the girl complained of pains in her back and head. Her throat was a little swollen and red.

The pulse throughout the attack remained quite uniform, and no elevation of temperature was noted. These attacks could be traced to no special cause, such as certain articles of food, cold, etc.

Her urine throughout the attack was light in color and contained neither casts nor albumen.

Case II.—Sophie L., aet. 27, a German by birth, married, applied for treatment on January 30th, 1890, complaining of the occurreuce of localized swellings in various parts of the body.

The patient's father is well, aged 56; her mother died of some lung trouble, probably phthisis.

With the exception of measles, when a child, and chills and fevers several years ago, she has never been ill; she has menstruated regu-

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larly, gives no history of any pelvic affection, had one child, which is living and in good health, and denies any miscarriage.

For some time previous to the occurrence of the affection to be described, she complained of feeling tired all the time, and suffered much from headaches. Eight weeks before admission she woke up in the middle of the night with a sensation of fulness and distension over her right eye, and noticed the presence of a swelling the size of a walnut almost completely closing this eye. In the morning the swelling had left her, but in the course of the day and every succeeding day similar swellings occurred, affecting her face, neck, hands, feet, and once her chest and abdomen, varying in size from a marble to a small orange, and preceded each time by a prickling, or itching sensation.

The 'lumps,' as she calls them, are usually red, and the skin over them glossy; there is no pain, unless the swellings occur in a position where pressure is naturally exerted upon them, as between two fingers, on the instep and similar localities. The rapidity with which they occur is astonishing, and one the size of a marble was observed to appear upon the dorsum of the left hand while in the examining room. These usually persist for several hours, and their disappearance is again accompanied by an itching sensation.

Difficulty in swallowing never occurred in her case, but at times a swelling would appear upon the mucous surface of the upper lip, everting the same completely and making the act of eating extremely painful.

Gastric symptoms were not noticed in this case, with the exception of occasional attacks of nausea on the days when she 'swelled' a great deal. At the time of her examination the patient was a well-nourished young woman, giving the impression of a nervous person, her eyes presenting the bright appearance so frequently observed in hysterical females; the pupils were somewhat dilated.

Examination of the thorax elicited nothing unusual.

Upon the dorsum of the left hand there was a swelling the size of a walnut at the junction of the middle and ring fingers, somewhat painful to pressure, and another smaller one on the right hand between the forefinger and thumb, no decided discoloration being noticeable.

Sudden barometric changes appeared to bring on an attack in her case, but no other causative influences could be traced.

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Her urine was pale in color and contained neither albumen nor casts.

During the five following weeks she seemed to improve under the administration of asafœtida and increasing doses of Fowler's solution, and when seen several months later she affirmed not having had a recurrence of the attacks.

CASE III.—Ida H., aet. 18, white.

As regards her family history it was ascertained that the mother had been for a number of years subject to fainting spells; one sister, aet. 15, had epilepsy; another younger sister has had her first fits lately.

When first seen patient complained of swelling of both hands and feet. Between the ages of 2 and 4 she had measles, scarlatina and diphtheria. Up to her 11th year she had attacks during which she would fall, lose consciousness, and become convulsed; since that time she has been subject to 'fainting' spells, in which she does not lose consciousness, however, knowing everything that is going on about her; she falls but is not convulsed. Such attacks have been coming on about every 3–4 weeks up to a year ago. Otherwise she had always been in good health up to the time when her present trouble began. This occurred four weeks ago, when upon waking one morning she found that her feet were swollen and painful when she attempted to walk. During the day this wore off, but toward evening the swelling returned, lasting throughout the night to disappear again the next morning. Two weeks ago her hands became similarly affected, and ever since the swelling has appeared every evening about 5 or 6 o'clock, reaching its maximum by 9 p. m. At this time the hands are very much swollen, the skin over them appearing tense, red and glossy; the natural depressions and irregularities of the palms are almost entirely obliterated, and movements practically impossible. As a rule the forearms are implicated, the swelling extending to near the elbow-joints. A general prickling sensation and feeling of distension is experienced and the finger-tips feel numb, pressure upon the parts exciting pain. The right arm, as a rule, is more affected than the left. At the same time the feet have become swollen, though to a less degree than the hands. This condition then persists until the next morning, when the feet return to their normal condition, while a slight degree of swelling may still be observed at noon in the hands.

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Symptoms pointing to laryngeal or oesophageal implications could not be ascertained. During the last two weeks, however, patient has had regular attacks of nausea, accompanied by headache, coming on regularly every evening and preceding the swelling by 1½–2 hours. Several times this has been followed by vomiting when the swelling was at its height. The ejected material is described as being like water. and has once been quite profuse. During the last week diarrhœa has also occurred, the stools, 4–5 a day, being watery in character, containing no blood or mucous. Abdominal pains have never been experienced.

Her menstruation has been regular, as a rule, but for a couple of months preceding the onset of the present trouble patient states that she menstruated every 2–2½ weeks; for the last month she has, however, been regular again.

Upon examination she was found to be a well-nourished, healthy-looking girl, weighing 163 lbs. Heart, lungs and abdomen appeared to be in a normal condition. Reflexes were not increased. At the time, 12 noon, scarcely any swelling could be made out, and no tenderness existed, yet patient affirms that the night preceding they had been very badly swollen, so that she could not bend her fingers. The next morning at 9 o'clock the fingers were somewhat swollen, and by 12 noon this had practically entirely disappeared.

Examination of the urine proved entirely negative as regards albumen and casts, its pale color, however, being quite striking.

During the following weeks the conditions described above continued. Then the swelling no longer attacked her feet or hands but only the index finger of the left hand, which would become swollen regularly every evening and the next morning be normal. Her face, however, now became affected and remained swollen for several days, after which it returned to its normal condition. During this week she had 3–4 watery stools a day, with great pain in the abdomen, usually preceding an evacuation. The vomiting did not re-appear during this time.

In the diagnosis of this affection the examination of the patient's urine becomes very important since cases occur which closely simulate the disease described but undoubtedly point to definite renal changes, as the following case will show:

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M. H., a little girl, aged 12, applied for treatment at the Johns Hopkins Hospital dispensary, complaining of oedema of the submental region, face and legs, which swelling occurred with astonishing regularity. Upon inquiry it was learned that the child had measles when 3 years old, but otherwise enjoyed perfect health. During the last two winters, however, she was subject to attacks in which the palmar surfaces of her fingers would swell up considerably every morning, the oedema disappearing about noon. Whenever the weather was cold the attacks occurred every day, and immersion of the fingers in cold water on such days made matters much worse, while bathing them in warm water had the opposite effect. Such an attack she had not experienced since last winter, and the oedema of which she complained at the time of her examination in June was of two weeks' standing, with daily remissions or intermissions, so to speak.

Her urine was examined every morning and evening for a time, and 2 per cent. of albumen discovered with an abundance of hyaline, epithelial and granular casts.

Similar cases have been repeatedly observed, and a diagnosis of angio-neurotic oedema has been corrected by the subsequent occurrence of Bright's disease in some form.

According to Strübing a slight amount of albumen may occur at the height of an attack, but large quantities, and especially the occurrence of casts in the patient's urine, will certainly throw grave doubts upon the correctness of the diagnosis.

The prognosis in these cases as far as life is concerned is good, since death only occurs in those rare cases in which sudden oedema of the glottis sets in, and a patient may pass through a long life with this affection as is seen in the case of Margaret A. of Osler's series, in whom the disease began at the age of 5 and continued until after her 50th year, and Allen of the same series illustrates this point still better for he was subject to such attacks from his 18th or 19th year up to his 90th. These cases illustrate at the same time the bad prognosis, as far as a complete cure is concerned, and the treatment really resolves itself into the careful avoidance of any known cause and the employment of tonic treatment and good food.

As to the pathology of the affection it is needless to say that next to nothing is known. We may speak of a condition of the blood-vessel walls, in which their permeability is increased, but this is

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certainly merely stating the matter in other words, for as long as our knowledge of the action of vaso-motor nerves upon the blood-vessels, and of the existence or non-existence of trophic nerves and centres is in its present embryonic state, it seems almost hopeless even to speculate upon the production of the so-called 'Angio-Neuroses.'

Vaso-motor influences alone, granting an increased permeability of the blood-vessel walls, seem insufficient to explain the phenomena, which are characteristic of the disease, and even the experiment of Ostromnoff, in which an oedema followed the stimulation of the peripheral stump of a cut lingual nerve, merely shows that nervous influences are capable of increasing the permeability of the blood-vessel walls, but does not prove by any means that this result is due to an effect upon the vaso-motor nerves. The same may be said of the experiments of Vulpian, Levascher and others.

Furthermore, Strübing's suggestion that peripheral stimuli of these nerves, while in a condition of increased irritability, may be the exciting factors in the production of the oedema, seems applicable, if at all, only in very few cases, and on very rare occasions, since the attacks occur without any traumatism, and are undoubtedly provoked by very much more subtle influences.

That the disease is one of the sympathetic system there is no doubt, for how otherwise can you explain the vomiting of such immense quantities of liquid, containing no serum albumen, as was seen in one of Strübing's cases? But to assume that vaso-motor influences alone are sufficient to bring about such results seems altogether unwarrantable. It is possible that they are concerned in the production of some of the symptoms, but future investigations into the existence and nature of a trophic regulative system will doubtless also show that it is here that we must look for an explanation of angio-neurotic oedema, and the angio-neuroses in general.

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HAEMATOMYELIA.

By AUG. HOCH, M. D.

It is a striking fact that while we so often meet with hemorrhage into the brain, hemorrhage into the spinal cord, not produced by any trauma, belongs to the rarest affections known in neuro-pathology. It has indeed been denied that we can ever speak of a primary spinal hemorrhage, the idea being that a softening of the tissue of the cord must always precede, and that the hemorrhage is thus only a secondary one. Monod, as early as 1830, doubted its existence; but the chief exponents of this view are Charcot, and after him Hayem.[1] This latter author has collected the cases, which up to his time were reported under the title of Haematomyelia, and has subjected them to a careful analysis. Among the thirty-one cases there was not one, Hayem thinks, in which we could reject the supposition that a change had taken place previous to the hemorrhage and so altered the tissue of the cord as to produce an extravasation of blood. Such a position, however, has been taken only by the just mentioned authors, while all the later ones, as, *e. g.*, Leyden, Eichhorst, Gowers, Erb, admit the existence of primary haematomyelia.

Previous to Hayem, Levier[2] in his monograph had given the histories of sixteen cases, fourteen of which are included in the thesis of Hayem. Since Hayem's time eleven more cases have been described, a collection of which we find in Berkley's paper on Syringomyelia.[3] I have been unable to find any further one reported with autopsy.

It would necessitate the writing of a monograph to review again all the cases and to discuss their exact nature, and it would in fact be an impossible task for, as Leyden[4] and Eichhorst[5] already have

[1] Hayem: Des hémorrhagies intrarachidiennes. Thèse des Paris, 1872.

[2] Levier: Beitrag zur Pathologie der Rückenmarksapoplexie. Inaugural Dissertation, Bern, 1864; also, Schweizerische Zeitschrift für Heilkunde, 1864, Band 3.

[3] Brain, 1889.

[4] Leyden: Ein Fall von Haematomyelie. Zeitschrift für Klin. Medicin, Band XIII, 1887, pp. 225–251.

[5] Eichhorst: Beiträge zur Lehre von der Apoplexia in der Rückenmarkssubstanz. Charité Annalen, Berlin, 1876, pp. 192–205.

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pointed out, we may be unable, even with a microscopical examination, to settle the question whether we have to deal with a primary softening and a secondary hemorrhage or with a primary hemorrhage and a secondary softening, analogous to the condition in the brain. Eichhorst and Gowers, therefore, admit, in opposition to Hayem, the possibility that some cases described as myelitis are really haematomyelia.

We see, therefore, that an anatomical diagnosis is not always possible, and that the clinical history with its different factors will be of great importance.

Undoubtedly, however, not a few cases described as such are not true examples of primary haematomyelia, but on the other hand cases like that of Goltdammer[1] *e. g.*, cannot be interpreted in any other way. Recently two cases have been reported by Krafft-Ebing,[2] diagnosed *intra vitam*, which we have every reason to call primary hemorrhage, also two cases by Sonnemann[3] from the clinic of Mendel.

It has been found by Charcot and Bouchard, and it is now a well-known fact, that the existence of miliary aneurisms in the cerebral arteries is a very common occurrence, so common that the above mentioned observers found them in seventy-seven consecutive cases of cerebral hemorrhage which they examined. This is, therefore, the most common cause of cerebral hemorrhage. We know that we find arterio-sclerosis in the spinal as well as in the cerebral arteries, but the soft pulpy brain with its comparatively large arteries and a high arterial pressure must be a more favorable situation for the formation of aneurisms than the denser spinal cord with smaller arteries and lower pressure,—not only for the formation of aneurisms but also for a hemorrhage to take place, sufficient to produce perceptible symptoms. Small hemorrhages are not unfrequently seen. But there are cases in which aneurisms similar to those in the brain have been described. Griesinger[4] reported a case where he found aneurismal

[1] Goltdammer: Beiträge zur Lehre der Spinal-apoplexie. Virch. Arch., Bd. LXVI, 1876.

[2] Krafft-Ebing: Zur Kenntniss der primären Rückenmarksblutung. Wiener Klinische Wochenschr., 1890, p. 939.

[3] Sonnemann: 2 Fälle von Haematomyelie. Inaugural Dissertation, Berlin, 1890.

[4] Griesinger: Archiv. für Heilkunde, Band 3, 1862, p. 174. Quot. by Levier (*loc. cit.*).

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dilatations in the cord of a boy who had died with tetanus, Hebold[1] in a case in which he attributed them to obstruction from venous thrombosis. Of special interest is the observation of Eichhorst,[2] who found in the case which he described as primary haematomyelia quite wide-spread aneurisms,—furthermore, the case of Wilkins[3] whose patient died with an acute spinal paralysis, regarded by the author as haematomyelitis, and where the existence of aneurismal dilatations in the grey matter of the upper dorsal region was noted.

While in the cases which we shall describe in this article we cannot entirely exclude the trauma, still we cannot class them under the head of the usual traumatic cases in which injury or concussion to the spine was immediately followed by a hemorrhage, since in one case six days, in the other three weeks, intervened between the accident and the paralysis.

The cases, therefore, seem to be of sufficient interest to be reported.

I am indebted to Professor Osler and Dr. Thomas, the chief of the Neurological Clinic, for the permission to report these cases. Case No. II has been reported by Dr. Thomas as "A case of Brown-Séquard's paralysis" before the medical society of the Johns Hopkins Hospital.

Case I.—K. I., aet. 40, an Irishman, laborer, came to the outpatient department on July 26th complaining of partial loss of power in his right arm and hand.

The account of the family history of the man is somewhat incomplete, since he has for many years not heard from his people. In previous years, and up to his present illness, he has had unusually good health and has not lost a day's work in the past fifteen years. He denies very positively any venereal infection, and says that he has for the past six years been a very moderate drinker, while previous to that he drank too much at times.

In the first week of June, 1890, while at work he was struck between his shoulders by a board which fell from the height of eight

[1] Hebold, Otto: Aneurismata der kleinsten Rückenmarksgefässe. Arch. f. Psych., Bd. 16, 1885, p. 813.

[2] Eichhorst, *loc. cit.*

[3] Wilkins: Case of spinal apoplexy. Montreal General Hospital Reports, Vol. 1, 1880, p. 111.

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or ten feet. He was knocked down but immediately got up again and felt no bad effects from the blow except some soreness on the following day at the spot where the board had struck. He went to work and felt well in every way up to about three weeks after this had happened. He came home one evening after a usual day's work, took his supper and felt quite well. He went on the street and sat down talking to a neighbor, only about half a square from his dwelling, when he was suddenly seized with a stabbing pain between his shoulders. He immediately arose and walked home, which he could do without assistance, noticing nothing at all in his legs. Soon he felt pain down the back, and when he lay down suffered from quite intense pain which had now also spread to both arms. The arms were drawn up across his chest and were tremulous. He remained in this condition for a few minutes and then completely lost power in his legs and partly in his arms, chiefly the right.

The time which elapsed between the onset of the pain and the paralysis was not more than eight or ten minutes. He did not lose consciousness at any time. Both arms could be moved at the shoulder-joints to some extent; the left was less affected than the right. He did not feel the people who came and rubbed him, nor could he feel a mustard plaster which was put upon his abdomen. He thinks he lost sensation completely on the body from about a line with the nipples down and in the arms. The physician ordered him a "sleeping powder," which had the desired effect, and the next morning he had no pain but felt numb in the places where the pain had been. His left leg could be moved a little and got better rapidly. About five days later he began to move the toes of the right foot, but the right leg improved more gradually than the left. Three days after the attack he could not hold anything in his hands. Both arms improved, the left more quickly and much more completely, the improvement in the right being chiefly in sensation. In general, sensation returned with motion. He could not pass his water for about two weeks, and the catheter had to be used. For some days also he had no stool, and took about the fourth day a purgative which acted. There was no priapism. As stated above we did not see the patient until July 26th, *i. e.*, about a month after the paralysis had come on. His condition then was as follows:

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Status Praesens.—Rather tall man. Face thin. Expression anxious. Muscles fairly well developed, little adiposa. Intelligence good. No abnormality about voice. Eye-balls freely movable. Left pupil larger than right, both react to light and accommodation. No trouble with vision, and in general with none of the special senses. Muscles of face act equally. Motions of head executed normally and with normal strength.

No tenderness and nothing abnormal about the spine. Trapezius (occipital portion) is more prominent on the right than on the left side. Shrugs shoulders and resists depression. Deltoids act on both sides and in all directions. Pectoralis somewhat subnormal on the right side. Rotates arm. Flexors of forearm act on both sides, somewhat weaker on the right. Extensors on the right very weak, on left strong. Motion about wrist on the right side is limited to the slightest flexion and extension. No motion in fingers. No contracture. On left side all motions of hand and fingers are possible, power is very subnormal. Muscles react to mechanical stimulation and upon being percussed a welt is formed at the place of contact.

I shall here only give an extract of the electrical examination, which was then not so complete as a later one, which will follow in extenso. In the left arm there was no electrical change found to faradism; with the galvanic current the muscles and nerves could be excited with about 2 M. A., K. C. C. with a corresponding A. C. C. In the right arm with the faradic the deltoid, biceps, triceps, supinat. longus, extens. radial., act well at about 41., as do the pector. and triceps—the triceps less than the others. Flexors do not act. Radial and ulnar nerve not obtained. With the galvanic the radialis, supinat. longus, extensor radial., biceps, deltoid, reacted quickly to a stimulation with 3 M. A., K. C. The triceps reacted less readily and rather sluggishly, and the A. C. C. was even somewhat larger than the K. C. C. The extensor communis, extensor ulnaris, extens. pollic., extens. indicis, showed slow contractions of a tetanoid character. The anode, however, being larger than the kathode. The flexors were excitable but reacted slowly; there were no qualitative changes in the reaction.

Roughly tested no sensory change was found. Heart and lungs were normal. The patient then left and did not return until Janu-

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ary 31st, when he was again examined, and the following notes were made:

Both legs look and measure equal. On a more careful examination no change is found in sensation to touch, temperature or pain, and faradism. Walks well. Pateller reflexes are active and equal on both sides, cremast. and plantar present. Electrically, all muscles and nerves of legs react well to galvanic and faradic, no diminution and no qualitative changes. Muscular strength good throughout in legs.

Body and arms: The right mammary region looks somewhat flattened, so that the intercostal spaces are more visible especially towards the sternum. The back looks symmetrical. Deltoids equal, and the muscular strength good in them. There is only slight difference in the pectoral. in favor of the left. The right upper arm is considerably thinner, and at the triceps seems especially flabby. Forearm on the right side is also thinner. The flexors especially seem flabby here. The right hand looks somewhat glossy. The surface over the knuckles and on the fingers more even than in a normal hand. The fingers are slightly bent, the hand extended. There is distinct atrophy of the thenar eminence. The left hand looks natural, except that the thenar of this side looks also flattened.

Measurements.	*Right.*	*Left.*
Wrist	14.8 cm.	16.0 cm.
Over supinat., with arm flexed at right angle	22.5 cm.	24.0 cm.
Greatest circumf. of upper arm	12.5 cm.	26.0 cm.
With arm extend. 13 cm., above ex. malleolus	19.0 cm.	21.0 cm.

Muscular strength of bicipites is fair and almost equal. Triceps is normal on the left side, very much subnormal on the right. The right hand can be flexed dorsally, but is held to the inside (radialis extensor). The left hand is raised higher and in median line, and the muscular strength in these muscles is good. Hardly any palmar flexion is possible in the right hand, while it is good in the left hand. No motion is possible in the fingers of the right hand; the thumb can be slightly separated from the other fingers. The motion in the fingers and hand of the left side is complete.

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Electrical Examination.—We would state here that the higher the number is, which is given as the point of the faradic stimulation, the stronger is the current, for the scale of our induction coils is arranged relatively, 100 being the strongest current.[1]

A medium-sized electrode was put on the sternum and for the different [Erb] electrode a button-electrode was used, and the smallest traction noted.

Faradic Current.

	RIGHT.	LEFT.
Biceps	13	17–22.
Deltoid, anterior part	17–22	17–22.
Deltoid, posterior part	17–22.	
Median nerve	28 (Pronat.)	28.
Ulnar nerve	Not obtained	28.
Radial nerve	Not obtained	22.
Triceps	17–22, slight and sluggish	22–28.
Supinator longus	17, good motion.	17–22.
Radialis extensor	28, good motion.	
Ulnaris extensor	Not obtained.	
Extensor communis	Not obtained.	
Interossei	Not obtained.	
Thenar	Not obtained	41–46, sluggish.
Hypothenar	Not obtained	Adduct. pollicis, 41–46.
Other small muscles	Not obtained.	
Flexors	Not obtained.	
Pronator	22.	

Pectoralis reacts less on right than left, the difference is mainly in the lower part of the pectoral muscle.

Galvanic.

Right Arm.—

Biceps, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Deltoid, anterior part, 3 M. A., K. C. C., 3 M. A., A. C. C. On stronger current, K. C. C. > A. C. C.
Lateral part, 1.5 M. A., K. C. C. < 4.5 M. A., A. C. C.
Posterior part, 2.5 M. A., K. C. C. < 3 M. A., A. C. C.
Triceps, 3 M. A., K. C. C. < 5 M. A., A. C. C.
Supinator, 2.5 M. A., K. C. C. < 4 M. A., A. C. C.
Ulnaris extensor, 2.5 M. A., K. C. C. sluggish < 3.5 A. C. C. sluggish.
Extensor communis, 4 M. A., K. C. C. slow, 4 M. A., A. C. C. somewhat quicker.

[1] The apparatus is described by Dr. Thomas in the *Bulletin* of the Hospital, July, 1890.

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Thenar, 1½ M. A., K. C. C. slow. < 3 M. A., A. C. C. slow.
Adductor pollicis, 1 M. A., K. C. C. slow. < 3 M. A., A. C. C. slow.
Opponens pollicis, 1 M. A., K. C. C, slow. < 2 M. A., A. C. C. slow.
Flexor profundus, 2 M. A., K. C. C. < 3 M. A., A. C. C. very slow.
Sublimus, 3 M. A., K. C. C. < 5 M. A., A. C. C. very slow.
Ulnar and radial nerve not obtained.

Left Arm.—

Radial nerve, 2.5 M. A., K. C. C. < 4.0 M. A., A. C. C.
Biceps, 1 M. A., K. C. C. < 3.5 M. A., A. C. C.
Triceps, 2 M. A., K. C. C. < 4 M. A., A. C. C.
Ulnar nerve, 1.5 M. A., K. C. C. < 2.5 M. A., A. C. C., 2.5 M. A., A. O. C.
Supinator, 1.5 M. A., K. C. C. < 2.5 M. A., A. C. C.
Radialis extensor, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Extensor communis, 2 M. A. K., C. C. < 3.5 M. A., A. C. C.
Extensor ulnaris, 2.5 M. A., K. C. C < 3.0 M. A., A. C. C.
Adductor pollicis, 2 M. A., K. C. C. < 4.5 M. A., A. C. C.
Extensor minimi digiti, 1 M. A., K. C. C. < 2.0 M. A., A. C. C.
Interrossei, 2 M. A., K. C. C. < 3 M. A., A. C. C.
Adductor pollicis, 2 M. A., K. C. C. < 2.5 M. A., A. C. C. rather slow.
Thenar, 1.5 M. A., K. C. C. < 2.0 M. A., A. C. C.

As regards the diagnosis of this case we have anticipated it in the title of this article. But if authorities like Leyden have expressed their opinion as to the impossibility of diagnosing *intra vitam* a haematomyelia it seems only natural that the reasons should be given why this seems the most probable diagnosis. A short recapitulation of the case will, therefore, I think, not be found out of place.

Patient was struck between his shoulders by a board falling from the height of eight to ten feet without producing any immediate effects save some soreness next day, which soon passed off and left him quite well. Three weeks later he was suddenly seized with acute pain between the shoulders radiating down the spine and into the arms, followed by a contracture of short duration in the arms, and within eight to ten minutes by complete sensory and motor paralysis from the arms down (with the exception of the higher-situated muscles of the arm). Retentio urinae et alvi. Improvement in the left leg the following day, comparatively rapid improvement also in the right leg and left arm, so that after three weeks there remained only paralysis of some of the muscles of the right arm, especially the forearm. On examination a month after, no sensory changes but paralysis with atrophy, showing the characteristic electrical changes of certain muscles of his right arm.

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We have, therefore, to deal with a sudden spinal paralysis which has destroyed the grey matter in a certain region of the cord. Before going into the question to what the lesion is due, let us localize it as far as it is possible, by finding out which muscles—as indicated by their impairment in motion and visible atrophy and by the results of the electrical examination—are interfered with in their nutrition, and we can then draw conclusions as to their trophic centres in the cord.

The muscles concerned are the following, classing them under two heads, namely, those which have completely lost their motion and show reaction of degeneration, and those which show only decrease in electrical excitability and some impairment in motion.

It will be seen, however, in the tables of the electrical examination that even those muscles which are the most degenerated do not fulfill all the requirements of the reaction of degeneration, as it has been given by Erb, but that we find the anode-closing contraction nowhere larger than the kathode-closing contraction, a condition which is not rarely seen.

I. *Muscles showing complete loss of motion and reaction of degeneration.* Extensor ulnaris, extensor communis; all the flexors; small muscles of the hand.

II. *Muscles showing decrease in electrical excitability and impairment of motion.* Lowest part of pect. major and triceps.

Now on what basis can we make an attempt to localize the lesion? A very important contribution to the spinal localization has been given in the valuable work of Thorburn.[1] From results of purely clinical observation he has mapped out a scheme of the distribution of the motor-nuclei, to which much importance is to be attributed, as the conclusions were drawn from a sufficient number of cases, observed and studied with care; and since they coincide in general with the anatomical investigations of Herringham[2] and the stimulation experiments of Yeo and Ferrier.[3]

According to Thorburn we have to place the lesion on a level with the VII and VIII cervical and I dorsal roots and partly upward in the VI cervical, and this as we will see seems to be the most plausible.

Thorburn's scheme is as follows:

[1] A Contribution to the Surgery of the Spinal Cord, by Wm. Thorburn. Philadelphia, Blakiston.

[2] Proceedings Royal Society, No. CCXLIII, 1886, p. 255. Quoted by Thorburn.

[3] Proceedings Royal Society, No. CCXII, p. 12. Quoted by Thorburn.

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VI. Subscap. pronat. teres. maj.; lateral dorsi. pectoralis maj.; triceps, serratus magn. VII. Extensor of wrist. VIII. Flexor of wrist. I. Dors: Interossei and other intrinsic muscles of hand.

The small muscles of the hand are by the three mentioned authors placed lowest down in the cord, the flexors and extensors in the region of the VII and VIII, by Ferrier, also in VI, V, IV, by Herringham in VI. The pectoralis could be stimulated by Ferrier from the VII and VIII, Herringham could trace fibres into VI, VII and VIII, while Thorburn refers its centre to the VI. It seems quite likely that the pectoralis has its centre in more than one segment of the cord. Both our cases, the one just reported and the one yet to follow, seem to point to this, for in both the lower part only was diseased, and we would conclude then that the centre for the lower part is situated lower (VI), while that for the upper is placed higher (V).

Another interesting point as regards localization is that we find the radialis extensor entirely preserved in our case. Thorburn does not speak of any nucleus for this muscle on a separate level from the others, while we find it especially mentioned by Herringham (VI and VII cervical). It is most probable that this muscle is represented higher up in the cord than the other extensors, for we have evidence of complete destruction of the anterior grey horns at the place where they are represented, while the extensor radialis remains quite intact. We see then that in general, according to the localization given by Thorburn, the muscles which we find diseased are represented in the cord in close connection, so that we can well understand how a hemorrhage in the grey substance, extending over somewhat more than three levels, can destroy their nuclei. So much then for localization.

Before going into the differential diagnosis, I propose to give the notes of the second case.

CASE II.—L. I., aged 21, railway brakeman, came to the surgical ward of the Hospital complaining of trouble in his hip, which proved to be a haematoma of considerable size. He was operated upon for this and left the Hospital well, as regards his surgical trouble, in the middle of January, 1891.

On October 18th, 1890, he was sent to the neurological clinic for examination because he complained of not being able to move his whole right side freely, the leg especially being somewhat stiff; he had also some disturbance in sensation in the left side.

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Anamnesis.—Patient had always been healthy and strong. Sometimes he has been drinking rather heavily. He denies any venereal infection. On the 18th of July patient met with an accident. While standing on a freight car he was knocked down and fell under the cars in the middle of the track, so that he was somewhat injured by two other box cars passing over him. He did not lose consciousness. The only wounds he had were on the forehead, and at different places on the scalp, and also on left foot, but his spine was uninjured and there was no sign of paralysis.

Six days after—he had meanwhile been brought to a hospital—he felt some pain in his right arm and leg, and did not sleep very much that night; when he awoke in the morning the right side, with the exception of the face, was completely powerless. His speech was not impaired. He slowly improved, the first sign of improvement being in the deltoid, then in the leg, while the fingers for some time were in a semi-flexed position, so that he neither could flex nor extend them entirely. Soon after the paralysis of motion he noticed also that on the sound side he could not feel as well when pinched as he could on the other. There was retention of urine and faeces for a few days and he had to be catheterized once.

Status Praesens.—December 12th. Strong-looking man. Walks with difficulty, on account of the haematoma on the left hip and apparently some weakness in the right leg. The hip is bandaged. Facial muscles act well and equally. Eyes are freely movable, normal. There is no abnormality seen on the spine and no tenderness. Shoulders look equal; the lower part of the right pectoral is somewhat flattened, as compared with the left. The triceps feels more flabby on the right side; the upper and lower arm somewhat thinner. The arm and hand held in natural position. The interosseal spaces are more marked on the right hand. The thenar eminence is quite flattened. Every motion seems possible.

Measurements.—	*Right.*	*Left.*
Upper arm, largest circumference.....	28½ cm.	29¼ cm.
Fore arm, largest circumference.....	28½ cm.	31½ cm.
Wrist.....	20¼ cm.	20½ cm.

Muscular Strength.—Deltoids good and equal. Pectorales: The left pectoral muscles are stronger. On contracting them the lower

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part of right pectoralis major is seen to be distinctly wasted. Extensors of the forearm: good on the left, much subnormal on the right. Flexors of forearm fair and equal on both sides. Dorsal flexion of the hand is possible to a greater extent in the left hand and the muscular strength there is somewhat better than in right. For the palmar flexors the same condition holds good. Grip on the left side is good, on the right subnormal. In the legs there is no apparent wasting, the right leg seems a little weaker than the left.

Reflexes.—Plantar not obtained on either side. Abdominal not obtained. Cremaster obtained on both sides; they are equal. Patellar reflexes are active on both sides, on the right exaggerated. Tendo Achillis reflex present on both sides, plus on the right. There is the most exquisite ankle-clonus on the right side, none on the left. Triceps: reflex well marked on the left, not obtained on the right. Periosteal reflex at wrist better marked on the left side.

Mechanical stimulation.—About the same on both sides.

Electrical Examination.

Faradic.

	LEFT.	RIGHT.
Nervus ulnaris	28	28
Nervus radialis	22–28	22–28
Nervus medianus	22	17–22
M. Biceps	17–22	13
" Triceps	22–28	22–28
" Deltoideus	22	22–28
" Extensor communis	22	28
" Extensor radialis	17–22	28
" Extensor ulnaris	17	22
" Extensor pollicis et indicis	22–28	28
" Extensor minimi digiti	28	—
" Supinator longus	22–28	22–28
" Flexor profundus	17–22	28–34
" Flexor ulnaris	28	28
" Flexor sublimus	28	28–34
" Pronator rad. ter	28	28–34
" Adductor pollicis	28	44–49
" Opponens pollicis	28–34	22–28
" Thenar	28	28–34
" Interossei	28	34
" Flexor minimi digiti	22	
" Adductor minimi digiti	28	

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As there was really no difference between the two sides as regards the strength of the current which produced the smallest contraction, we concluded to stimulate all the muscles with a fairly strong faradic current, and note the difference in the strength of the contractions, which in some of the muscles was quite apparent. The result was as follows: With a current of 49, bicipites react equally. The reaction of the ulnar nerve is less strong and less quick and less tetanic on the right side; extensor communis less active on right side; supinatores almost equal; extensores radial. equal; extensores ulnares almost equal; pronatores equal; flexor. profund., flexor. carpi radial., flexor. sublim. equal on both sides; flexor carpi ulnaris perhaps a little more sluggish on the right side; the triceps more sluggish and less tetanic on right side; pectoralis less on right side; opponens and adductor pollicis decidedly less on right side.

Galvanic Current.

Left Arm.—

Ulnar nerve, $2\frac{1}{2}$ M. A., K. C. C. < 6 M. A., A. C. C., A. O. C., 8.
Radial nerve, 2 M. A., K. C. C. < $3\frac{1}{2}$ M. A., A. O. C., A. C. C., + 8.
Median nerve, $1\frac{1}{2}$ M. A., K. C. C. < 3 M. A., A. O. C., A. C. C., + 8.
M. Deltoid, ant. part, 4 M. A., K. C. C. > $3\frac{1}{2}$ M. A., A. C. C.
mid. part, $5\frac{1}{2}$ M. A., K. C. C. > 5 M. A., A. C. C.
post. part, 6 M. A., K. C. C. > $5\frac{1}{2}$ M. A., A. C. C. } quick.
" Triceps, $2\frac{3}{4}$ M. A., K. C. C. < 5 M. A., A. C. C.
" Biceps, $1\frac{1}{2}$ M. A., K. C. C. < $1\frac{3}{4}$ M. A., A. C. C.
" Supinator, 2 M. A., K. C. C. < 3 M. A., A. C. C.
" Extensor radialis, $2\frac{1}{2}$ M. A., K. C. C. < $4\frac{1}{2}$ M. A., A. C. C.
" Extensor ulnaris, $2\frac{1}{2}$ M. A., K. C. C. < 4 M. A., A. C. C.
" Extensor communis, 4 M. A., K. C. C. — 4 M. A., A. C. C.
" Extensor pollicis, $4\frac{1}{2}$ M. A., K. C. C. < 6 M. A., A. C. C. and A. O. C.
" Extensor indicis, 2 M. A., K. C. C. < $2\frac{1}{2}$ M. A., A. C. C.
" Interossei, 2 M. A., K. C. C. < $3\frac{1}{2}$ M. A., A. C. C.
" Thenar, $4\frac{1}{2}$ M. A., K. C. C. > $3\frac{1}{2}$ M. A., A. C. C.
" Flexor carpi ulnaris, $2\frac{1}{2}$ M. A., K. C. C. < 5 M. A., A. C. C.
" Flexor profundus, $2\frac{1}{2}$ M. A., K. C. C. < $3\frac{1}{2}$ M. A., A. C. C.
" Flexor sublimis (II, III), 2 M. A., K. C. C. < 5 M. A., A. C. C.
" Flexor sublimis (ind. and little), 2 M. A., K. C. C. < 5 M. A., A. C. C.
" Adductor pollicis, 2 M. A., K. C. C. < 6 M. A., A. C. C.
" Opponens, 3 M. A., K. C. C. < 6 M. A., A. C. C.
" Flexor carpi radialis, 3 M. A., K. C. C. < 5 M. A., A. C. C. All the contractions are quick, not sluggish.

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Right Arm.—

Ulnar nerve, 1½ M. A., K. C. C. < 5 M. A., A. C. C.
Radial nerve, 2½ M. A., K. C. C. < 4½ M. A., A. C. C.
Median nerve, 2½ M. A., K. C. C. < 2¾ M. A., A. O. C., 8½ M. A., A. C. C.
M. Extensor radialis, 3½ M. A., K. C. C. < 5½ M. A., A. C. C.
" Supinator, 3 M. A., K. C. C. — 3 M. A., A. C. C.
" Extensor ulnaris, 2 M. A., K. C. C. < 5 M. A., A. C. C.
" Extensor communis, 3 M. A., K. C. C. — 3 M. A., A. C. C.
" Extensor indicis, 2½ M. A., K. C. C. < 4½ M. A., A. C. C.
" Extensor pollicis, 2½ M. A., K. C. C. < 4½ M. A., A. C. C.
" Deltoideus, 2–3½ M. A., K. C. C. < 4½–6 M. A., A. C. C.
" Flexor ulnar, 2½ M. A., K. C. C. < A. C. C., not obtained.
" Pronator, 2 M. A., K. C. C. < 6 M. A., A. C. C.
" Flexor profundus, 2 M. A., K. C. C. < 4 M. A., A. C. C.
" Flexor sublimis, 2½ M. A., K. C. C. < 6 M. A., A. C. C.
" Flexor pollicis, 3 M. A., K. C. C. < 6 M. A., A. C. C.
" Opponens, 3½ M. A., K. C. C. < 5 M. A., A. C. C.
" Interossei, 3 M. A., K. C. C. < 4 M. A., A. C. C., slow.
" Thenar, 3½ M. A., K. C. C. < 6 M. A., A. C. C., slow.
" Adductor pollicis, 5½ M. A., K. C. C. < 8 + M. A., A. C. C., slow.

Sensation.—As the patient himself had noticed, there was a distinct difference in pain sensation on the side not affected by the motor paralysis. On the whole leg analgesia existed, and a pin was only felt when it penetrated quite deeply into the skin ; this involved half of the scrotum and half of the penis. On the body this was less noticeable, and was more marked in the lower parts and closer to the spinal column. Upwards this dulling of the sensation to pain extended to about the third rib in front, and about the same height on the back and down the back of the arm. It reached the median line anteriorly and posteriorly.

From about the middle of January there was no perceptible difference on the two arms. Over the same area and about similarly distributed as regards intensity, there was loss of temperature sense, this being abolished even to very marked differences of hot and cold, becoming less dull, however, in about the same area as the pain sensation did. *As to touch there was not the slightest dulling anywhere on the body.* A zone of hyperæsthesia could not be made out at any time while the patient was under observation, neither could any change in the muscular sense be determined.

To sum up then, *the patient fell from a train, sustained several injuries, but no perceptible one to the spine. No trace of paralysis for*

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six days; at that time some pain in right extremities, and after some hours' sleep complete paralysis in the right leg and arm. On examination, three months after, weakness of triceps, pectoralis, flexors and extensors and small muscles of the hand of right arm, with decrease in electrical excitability in some of these muscles (triceps, pectoralis, some of the small muscles, extensor communis); also some visible atrophy; furthermore, stiffness in the right leg; no atrophy or electrical changes in muscles of leg; loss of the sense of temperature and pain on the whole non-paralysed left side.

We have here, therefore, a case of Brown-Sequard's paralysis, resembling the picture of a syringomeyelia.

The diseases which we have to think of in our differential diagnosis we shall consider now. We can readily in both cases exclude a cerebral lesion, but the spinal affections other than haematomyelia which can give rise to such an acute paralysis are: 1. Hemorrhage into the spinal canal outside of the cord or haematorrhachis. 2. Acute myelitis. 3. Acute ischaemic myelomalacia.

In Case II, it is not difficult to exclude haematorrhachis, for we cannot imagine that by such a hemorrhage sensation alone on one side and motion alone on the other should be affected. In the first case haematorrhachis might have suggested itself at the beginning, but the course which the disease took, and the nutritive disturbances in the muscles can hardly be brought into accord with that diagnosis, even if we assume the possibility of a clot forming between the dura and the bony canal; in that case we would also rather expect to find changes in the other arm as well. Besides the examples of such a localized clot are very few.[1]

Myelitis.—The strongest point against myelitis in our cases, especially Case I, is certainly the very acute onset. A paralysis which, without any prodromes save an acute pain lasting for a short time, and transient contractures, destroys *within a few minutes* motion and sensation from the arms down, cannot be explained in any other way than by hemorrhage. In Case II, while the onset was certainly very acute, we cannot say positively how long it took from the first symptoms to the complete paralysis; but the paralysis did not spread

[1] Moulton (Thèse de Strassbourg, 1887) quotes a case of this nature, reported by Laennec.

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after the patient had awakened. An acute anterior polio-myelitis, of which we certainly have to think in Case I, while often very acute, never begins and reaches its highest development in so short a time. An anterior polio-myelitis is furthermore never accompanied with sensory changes, which have been quite marked in our case, and lastly it is accompanied by fever and general disturbances.

The quick regression of some of the symptoms which we found in Case I were also much in favor of hemorrhage. Symptoms of paralysis which disappear in such a short time cannot be due to inflammation and we have to look for another cause, and pressure naturally suggests itself. Krafft-Ebing,[1] in a somewhat similar case, in which, however, the hemorrhage was into the posterior horn, explained the motor paralysis by shock. The term "shock" does not convey any definite idea however, and in fact neither does pressure, but the symptoms seem to be to some extent explained by what is usually called pressure. No doubt infiltration and secondary inflammation play some part. Anterior polio-myelitis also may produce symptoms which by some are supposed to be due to pressure, owing to extravasation of blood. We would expect them in this disease, however, to come on later and to be less extensive.

Therefore, we can almost with certainty exclude myelitis in Case I. While we cannot, it is true, be so sure in Case II, still I think that with the acute onset the limitation of the apparent destruction is in favor of hemorrhage.

Here I may mention the three well-known cases described as unilateral hemorrhage, with symptoms of Brown-Séquard's paralysis, reported by Monod, Oré, Brechet.[2] Also a case which more closely resembles our Case II. It is one reported by Rosenbach and Schtscherbach,[3] under the name of syringomyelia. It is true the case resembles very much the complex of symptoms presented by syringomyelia, as in fact our case does, but considering that the patient had

[1] *Loc. cit.*

[2] Extracts of all three cases are given by Levier, *loc. cit.*; all three typical cases of Brown-Séquard's paralysis.

[3] Zur Casuistik der Syringomyelie, by Rosenbach and Schtscherbach, in St. Petersburg. Neurolog. Centralblatt (Mendel), 15 April, 1890, p. 226. Patient fell down several steps on his back; could not raise right foot; the hand had lost the motion of the fingers; gradual improvement. Half a year later atrophy of some muscles of arm; on unparalyzed side analgesia and loss of temperature sense.

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had a fall, and immediately after it the symptoms had developed, it seems more natural to attribute these symptoms to a hemorrhage, just as in our patient, than to an acute exacerbation of a previously existing process which had not been noticed by the patient.

Acute ischaemic myelomalacia is one of the rarest affections and little is known about it. Leyden is said to have reported two cases in which he had found emboli in spinal arteries in the course of an acute ulcerative endocarditis. We can imagine how an embolism or thrombosis can give rise to some localized nutritive disturbances, such as Klebs has lately described,[1] but not to such a limited acute softening, for we have in the cord a different vascular condition from the brain and not a system of end-arteries. But even if this could happen, the softening ought to be confined to the area in which destruction remained, and we would have no explanation for the extensive pressure symptoms in Case I. Cases have been reported in which complete myelomalacia followed cutting off of the blood supply in the abdominal aorta.

We have yet to consider the influence which the accident had in the production of the hemorrhage in these two cases. It seems hardly conceivable that in both the concussion which preceded—even though there intervened a considerable time between it and the spinal symptoms—should be in no causal relation with the disease. Far from endeavoring to give a definite explanation of this *nexus causalis*, which would in fact be impossible, I think it will be interesting to consider the spinal lesions produced by a concussion. It is not uncommon to find a hemorrhage following immediately either a fall or blow on the back. Such cases have often been described, and we find some good examples in the above-mentioned work of Thorburn; but the changes seem more frequently to be of a different nature. Symptoms of a subacute myelitis, or it has been claimed, of a distinct system-disease (tabes), may appear, or a paralysis occurs which is not explained by any appreciable anatomical changes. What are the early changes which are produced by concussion? Schmaus,[2] in endeavoring to answer this question has studied it experimentally and

[1] Klebs: Ueber Landry'sche Paralyse. Deutsche Medicin. Wochenschrift, 15 Januar. 1891.

[2] Dr. Hans Schmaus: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung. Virchow's Archiv, Band 122, pp. 328 and 470.

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shown that the first anatomically appreciable changes consist in death of the axis-cylinders. In the anterior horns was found an indefinite granular degeneration. Hemorrhage and changes in the vessels did not occur, but he observed circumscribed foci of softening with cavity formation, and also gliosis with cavity formation. (Each of these changes in one experiment; on the animal with extensive gliosis the autopsy was made twelve days after onset of disease).

There is still another class of which Gowers speaks, where we have no symptoms immediately after the concussion, but where later signs of subacute myelitis appear. The last class then would be formed by cases such as ours,[1] in which there are no symptoms for a longer or shorter period of time, and then a sudden onset. The cases are undoubtedly rare, and it is difficult to interpret them. Had any of the above-mentioned changes taken place, we would have expected them to be manifested by symptoms. That we find gliosis produced by concussion cannot explain Case II—although it resembles the clinical picture of syringomyelia—for the same reason. Whether we then have a change produced chiefly in the vessels without any parenchymatous alterations sufficient to cause symptoms can only be a supposition. This delay in the effect of the concussion is certainly not the least interesting feature of our cases.

[1] There are two cases reported under the title of Haematomyelia, which are of interest in this connection. The case of Peddie (Monthly Journal of Medical Science. of July, 1846, and June, 1847, pp. 819–833), in which ten days after spinal concussion an acute paralysis came on. The case of McMunn (Dublin Journal of Medical Science, Vol. 59, 1880, pp. 182–189), in which two days after spinal concussion the acute paralysis came on. In both cases, no symptoms in the intervening time.

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A CASE OF CEREBRO-SPINAL SYPHILIS, WITH AN UNUSUAL LESION IN THE SPINAL CORD.

By HENRY M. THOMAS, M. D.

CLINICAL SUMMARY.—*Male, æt.* 33. *No syphilitic history. Paralysis of right sixth cranial nerve, accompanied by intense headache in January, relieved by treatment. In May, headache and paralysis of the left fourth nerve. In November, paralysis of the left third and fourth nerves. Weakness of the muscles on the right side of the body, with slight sensory changes. Increasing coma. Death.*

ANATOMICAL SUMMARY.—*Syphilitic orchitis. Syphilitic endarteritis (gummatous) of cerebral arteries. Gumma on left third nerve, involving left crus. Gummata on left fourth, right sixth, ninth, and twelfth nerves and in brain. Gumma on anterior roots of third cervical nerves. Meningitis of cord. Poliomyelitis of lumbar enlargement. Hyaline degeneration in the walls of the small arteries.*

The patient was first seen at his house two weeks before his death, when his mental condition was such as to make it impossible to obtain from him any reliable history of the cause or beginning of his illness.

His friends were able to tell but little about his previous history, so that the notes which were taken on his admission to the Hospital are incomplete. However, through the kindness of Dr. Hiram Woods, who saw the patient several times, and who has lent me his notes, I have been able to add some important facts which explain in an interesting way the lack of agreement between the symptoms noticed during life and the condition found at autopsy.

This case seems to me again to emphasize strongly the importance of a full and accurate history of an illness, if we are to hope for a diagnosis more than partially correct.

The history which was obtained at the Hospital from his friends was as follows:

J. K., æt. 33, driver, admitted to the Hospital November 14, 1889.

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Up to the beginning of the present trouble the patient has been a strong, healthy man. As a younger man he led a very irregular life, but his friends do not know whether he contracted syphilis or not. A little more than a year ago he began to complain of a very severe pain in his head, in the hope of relieving which he had his teeth drawn. After this operation his mouth became very sore. His headache has persisted in greater or less intensity ever since, often being so severe as to prevent his sleeping at night, and to require the use of opiates.

Soon after his head had begun to ache, he complained of seeing double, and went for treatment to the Presbyterian Eye and Ear Hospital. His friends think his right eye was affected at that time.

His left eye-lid has been drooping for several months.

From time to time the patient has complained that the right side of his face felt numb. The weakness in the muscles of his right arm and leg has been noticed only during the last few weeks, and was discovered by his physician.

The patient had to give up work five weeks ago, and has been in bed three weeks. His mind became evidently affected on the 13th of November.[1]

Present condition.—Intelligence is clouded. He is able to obey commands but his answers are unreliable, as he often gives different answers to the same question.

[1] Abstract from Dr. Hiram Woods' notes:

J. K. was seen first on January 28th, 1889. He came complaining of intense pain in the head and double vision. An examination revealed a paralysis of the right external rectus.

Vision, $\frac{20}{20}$; eye-grounds normal.

Patient acknowledged having had gonorrhoea but denied syphilis, and no history of secondary symptoms could be obtained.

Iodide of potassium was given three times a day in 40 grs. doses, and passive motion was applied to the weak muscle. Under this treatment he rapidly improved, the muscle recovering completely.

In May of the same year he was again seen. He complained this time as before of intense pain in the head and double vision. His double vision troubled him when he looked down. Upon examination all the muscles of the right eye were found to act normally. Tests by prisms revealed a weakness of the superior oblique muscle of the left eye.

Potassium iodide was ordered, but the patient was lost sight of, and so the result of the treatment cannot be stated.

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Eyes.[1]—The left pupil is dilated; the right is of normal size. The left pupil does not react to light, and the right only sluggishly. The left eye-lid droops and he is unable to raise it. He cannot move the left eye either to the right, up or down; he is able to move it toward the left.

Face.—The muscles which contract and raise the eye-brows, and those which close the eyes act well and equally on both sides of the face. When attempting to expose his upper teeth the muscles on the left side of the mouth act slightly more than those on the right. He opens his mouth with difficulty, and in doing so the lower jaw is moved slightly to the right. The right side of the tongue is higher than the left. The tongue is protruded towards the right.

Sensation is practically normal, perhaps a little dull on the right side.

Hearing is sub-normal on the right side.

Arms.—Muscular strength of right arm is decidedly below that of left. The deep reflexes are exaggerated on both sides.

Legs.—The right leg is markedly weaker than the left. The patellar clonus and ankle clonus are well marked on both sides, and the plantar and cremasteric reflexes are also very active.

The clinical diagnosis was of a tumor of the left crus, involving the left third, and possibly the fourth nerve. The growth was thought to be syphilitic. He was put upon large doses of iodide of potassium with mercurial inunctions.

The patient remained in practically the same condition except that he became more and more unconscious.

On November 25th, the note is: "Patient in a semi-comatose condition, cannot be easily roused, passed a very restless night. Stiffness in right arm very marked. Patellar clonus very much diminished, so also the ankle clonus. The knee-jerks are still active, especially on the right side."

For thirty-six hours before death the patient lay in a comatose condition; mucus plugged up the respiratory passages; respiration became very rapid; pulse not to be counted. He died quietly about 2 a. m., November 28th.

[1] An opthalmoscopic examination was made, and the eye-grounds were found to be normal; but as there is no note to that effect in the Hospital history, and I have to depend upon my recollection of this point, I have omitted it from the text.

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Autopsy.—Seven hours after death. Body large, strongly built, well nourished. Rigor mortis strongly marked in all the extremities. On the dorsum of the glans penis, just above the corona, there is a small depressed cicatrix. Skin over the body is smooth and presents no evidence of a former eruption.

Abdomen.—The abdominal cavity contains a few cc. of clear fluid. Peritoneum normal.

The liver, spleen, kidneys, stomach and intestines are all apparently normal. Both testicles are firm, indurated, and contain a fine diffuse growth of connective tissue.

Thorax.—Praecordial space uncovered by lung 8 × 6 cm. Both lungs adherent to pleura by slight adhesions; in left pleural cavity a few cc. of clear fluid; in right same amount, somewhat cloudy.

Both layers of pericardium smooth. Heart flaccid; on both sides of heart fluid blood mixed with dark coagula.

Myocardium pale. Left ventricle 12 mm. thick, right 3 mm. Aorta, above valves, 73 mm. wide. Valves normal. Coronary arteries wide, with here and there slight thickening of the intima. Aorta contained a few whitish non-calcified patches.

In the posterior portion of the lower lobe of left lung a few areas of croupous pneumonia. Lung elsewhere slightly oedematous. Mucous membrane of bronchi injected; pus squeezed from small bronchi in any place in lung.

Entire posterior portion of lower lobe of right lung, and small portion of middle lobe in a state of gray pneumonic consolidation. Bronchi red, and injected. Pus in small bronchi everywhere.

Brain.—Skull-cap thin, dura not adherent, vessels of dura full, longitudinal sinus contains large clots; the veins of the pia entering the longitudinal sinus much distended.

Base.—Arachnoid clear, olfactory bulbs and nerves normal; optic nerves and chiasm uninvolved; infundibulum and pituitary body normal; the right third nerve lies free—a little reddish at its point of emergence around the crus.

The left third nerve is involved at its point of emergence in an ovoid gummous mass, which lies between the left cerebral artery and the anterior margin of the pons and the inner margin of the left crus. The gumma appears to be really in the nerve itself.

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The left fourth nerve as it passes around the crus is involved in a small gumma the size of a split pea; on the right side the fourth nerve is free.

Fifth nerve, left side, is uninvolved; right side there is little matting and adhesion to the dura.

Sixth nerve on left side normal; on right side the first centimeter of the nerve, after emergence behind the pons, is involved in an ovoid gummatous growth. Running transversely across this, and adherent to it, is a branch of the basilar artery.

Seventh pair on both sides uninvolved.

The pneumogastric on the left side free; on the right side the lower two or three roots are involved in a small gumma. The left spinal accessory and hypoglossal normal.

On the right olivary body, and involving the upper filaments of hypoglossal, there is an irregular gumma 12 × 6 mm.; it is attached to one of the posterior cerebellar arteries, a branch of the right vertebral.

Arteries.—The carotid on both sides normal; the right anterior cerebral as it crosses the inner root of the olfactory nerve is hard and firm, and surrounded by slightly thickened meningeal tissue. At the anterior communicating there is a small nodular gumma; the meninges are here adherent, and there is a considerable area of gummous infiltration, chiefly involving the inner aspect of the first frontal gyrus. The anterior cerebrals beyond this point are small; they contain blood; the left anterior cerebral from the carotid to the anterior communicating is closely adherent and surrounded by thickened gumma tissue.

The posterior communicating on both sides normal; the right is larger than the left.

The right posterior cerebral is free; the left at its emergence from the basilar is opaque-white in color, and is adherent to the gumma on the third nerve.

The basilar presents three ovoid dilatations due to the presence of gummata in its walls.

The left vertebral is free; the right is involved in a small gumma just as it passes along the side of the olivary body.

On one of the cerebellar branches of the basilar on the right side is a small gummatous nodule. The left crus is softened at its outer and under aspect; the pons and medulla appear normal.

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The convolutions at the base present no special anomalies.

The fifth ventricle is dilated; the hemispheres are normal, white and gray matter are firm and healthy looking; the ventricles are not dilated.

Cord.—Meninges of cord of ordinary thickness. Cord itself firm. On the anterior roots of third cervical nerve on right side, about 1 cm. from the cord, a small gummatous tumor.

Anatomical diagnosis.—Croupous pneumonia of both lungs, more marked on right side. Acute bronchitis. Syphilitic orchitis. Syph-

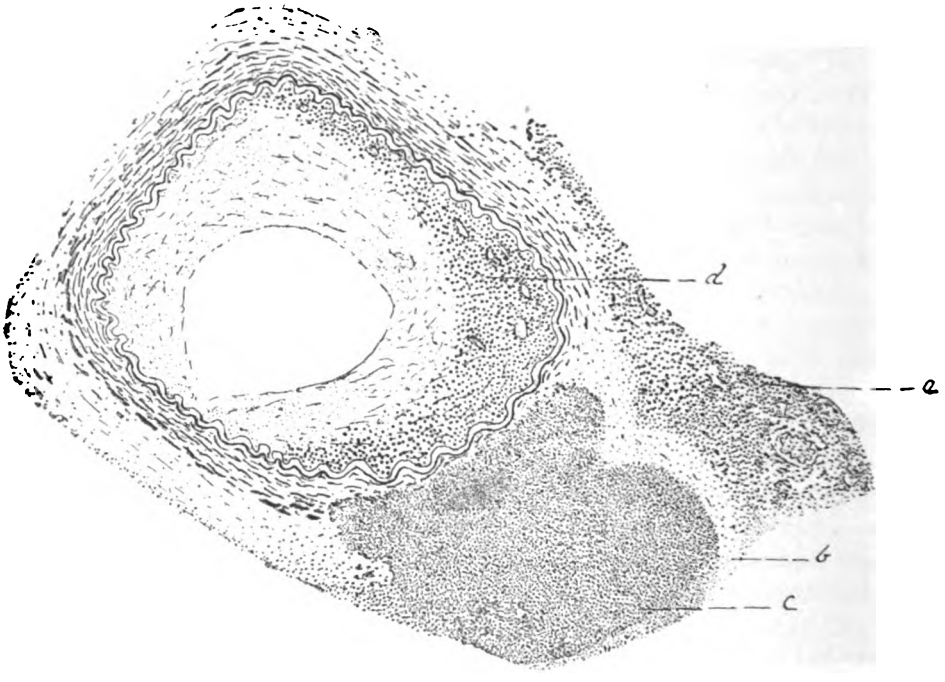

FIG. I.

ilitic endarteritis (gummatous) of cerebral arteries. Gummata on cranial nerves and in brain. Gumma of third cervical nerve. Anterior poliomyelitis in upper lumbar cord.

Microscopical Examination.—The brain and spinal cord were hardened in Müller's fluid, and many microscopical sections were prepared and studied. This work was done in the Pathological Laboratory of the Johns Hopkins University, and I am indebted to Dr. Welch and Dr. Councilman for their advice and assistance.

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Arteries.—Sections of the basilar artery (Fig. 1) at the site of the enlargements noticed at the time of autopsy, show the following condition. The nodules in the wall of the artery are seen to be composed of three, more or less, distinct zones. The outermost layer (*a*) is made up of a great number of small round cells, whose nuclei stain deeply and well with haematoxylin. This layer is rich in blood vessels, which are filled with red blood corpuscles. Here and there one sees a larger blood vessel, the wall of which is completely infiltrated with these small cells, and whose lumen is obliterated. In the second layer (*b*) the cells are not so plentiful, their nuclei stain well, and one is able to distinguish the formation of fibrous tissue. The third and innermost layer (*c*) is composed of necrotic tissue; in it there are no well-stained nuclei, but merely the remains of these. This tissue appears firm and fibrous, and contains the remains of obliterated blood vessels.

This new growth occupies for the most part the adventitia of the artery, but also encroaches in places on the muscular coat.

The infiltration of the small round cells which make up the outer coat is not confined to the nodule itself, but extends in the adventitia to a greater or less extent all around the artery. The muscular coat, except where it is invaded by the new growth, seems to be normal. The elastic lamella is intact, but the internal coat is very extensively thickened. This thickening is made up of new-formed fibrous tissue, and encroaches upon the lumen of the artery very materially. At places (Fig. 1, *d*) there is an infiltration of small cells into the thickened intima. Where this infiltration occurs, it occupies the portion nearest the lamella, and in it we see numerous new-formed blood vessels.

Sections through the other arteries at the base of the brain show them to be all more or less diseased. Where their walls are not the seat of actual gummatous growths, the adventitia is usually infiltrated with small round cells, and the intima is the seat of an increase of connective tissue. The muscular coat usually appears normal, but it is in places thinned, and here its nuclei have lost their characteristic appearance. This change usually extends only part way round the artery, and we find that the greatest thickening of the inner coat corresponds accurately to the degenerated part of the muscular coat. These changes vary greatly in different arteries; both internal caro-

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tids, just as they enter the base of the skull, are affected, the right much more than the left.

Nerves.—At the autopsy it was found that the left third, the left fourth, the right sixth and some roots of the ninth and twelfth nerves on the right side were involved in small tumors. Sections of these tumors prove them to have the typical structure of gummata (Figs. III, IV, V). Sections of the left third and fourth nerves

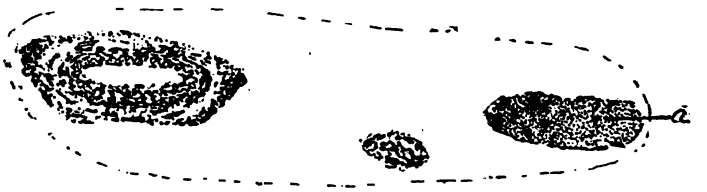

FIG. II.

(Fig. II, *a*) beyond the new growth show an almost complete degeneration of the nervous elements, and a substitution for them of a connective tissue rich in cells. The degeneration of these nerves is well seen in sections prepared from the tissues which occupy the back of the orbit on the left side. On these preparations we have sections of the optic nerve, of the nerves which supply the muscles of the eye-ball, and sections of those muscles themselves. The degenerated

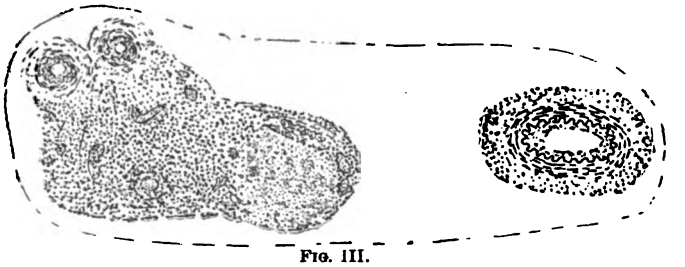

FIG. III.

nerves are easily distinguished by the contrast which they make to the healthy branches of the sixth. It is interesting to notice the well-marked atrophy of certain muscles, presumedly those to which the diseased nerves are distributed.

The optic nerves are normal on both sides.

The gummata on these cranial nerves are all connected with the walls of small arteries. This is well illustrated by the fourth nerve,

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parallel to which runs an artery, which becomes closely adherent to the gummatous mass. The sections of this nerve and artery (Figs. II, III, IV) taken from different levels illustrate this connection, and it is also well seen in the series of sections of the gumma on the

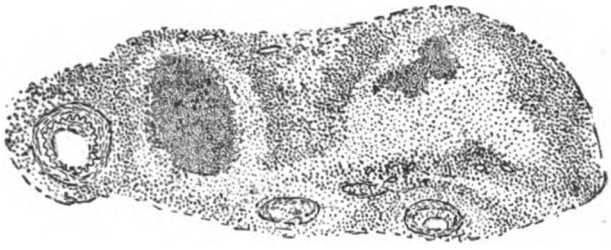

FIG. IV.

right sixth nerve (Fig. V), which show the artery running over this gumma, becoming obliterated and lost in the tumor mass. Sections of the right sixth nerve beyond the gumma show that this nerve, although not normal, still contains quite a number of well-preserved

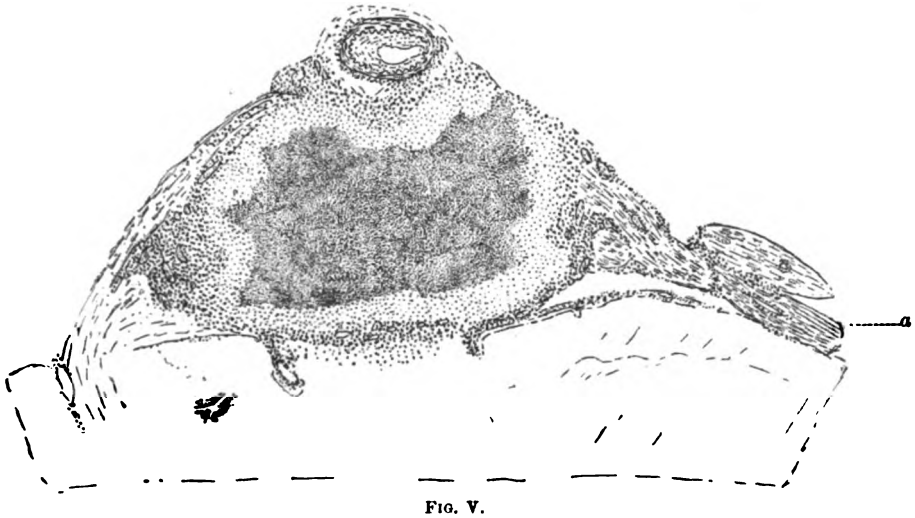

FIG. V.

nerve fibres contrasting strongly with corresponding sections from the third and fourth nerves. As compared with sections of the left sixth nerve it is smaller, contains more connective tissue and fewer nerve

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fibres. Sections (Fig. v) of this gumma, prepared so as to cut the nerve longitudinally, show its relations in an interesting way. The tumor seems to have grown in the nerve itself, and to lie between the artery and the surface of the pons. As has been said above, the artery is lost in the tumor mass. The superficial layers of the pons have been invaded by an infiltration of small cells, and its nervous

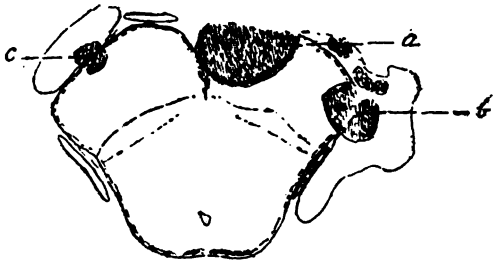

FIG. VI.

elements have been destroyed. Nerve fibres are seen entering the tumor at its lower border, and some sections show bundles of nerve fibres cut longitudinally emerging from its upper boundary (Fig. v, *a*). These sections stained in Weigert's haematoxylin demonstrate that these fibres still retain their myelin sheath. The neurilemma which separates these bundles of nerve fibres is infiltrated

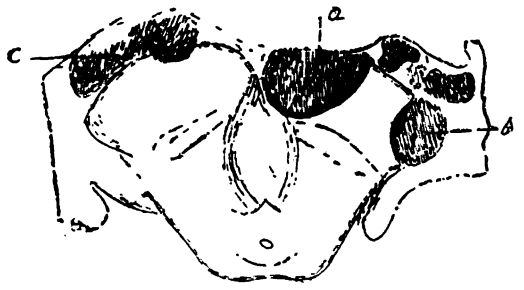

FIG. VII.

with small cells, and the blood vessels which run in them are also surrounded by these cells.

The gumma involving the third nerve also extends into the substance of the left crus. Sections cut at different levels through the crura (Figs. VI, VII, VIII, IX) show that this tumor (Figs. VI, VII, *a*)

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involves about the inner third of the cross section of the left crus and extends anteriorly for a little more than one centimeter. These sections show that this part of the brain is also invaded by numerous other gummatous growths. In the section nearest to the pons (Fig. VI, *b*) there is on the left side, besides the gumma described above, a smaller tumor situated partly in the crus involving its outer angle and partly in the structures surrounding it. There are also other still smaller gummata in the surrounding tissue. On the right side the crus is encroached upon, at its periphery, by a small gummatous mass (Fig. VI, *c*). Towards the brain we find the tumor associated with the left third nerve becoming smaller and disappearing (Figs. VII, VIII, *a*), while the tumor noticed at the angle of the left crus, together with those in the surrounding tissue, become larger and

FIG. VIII.

seem to coalesce, forming an irregular gummatous mass, lying for the most part outside of the crus, but infiltrating its periphery to a greater or less extent (Fig. VIII, *b*). Still higher up this gumma becomes more and more confined within the limits of the crus, occupying about its middle third (Fig. IX, *b*). The gumma on the right side increases as we ascend, involving the right crus to quite a considerable extent; it is situated for the most part in the middle third (Figs. VII, VIII, *c*).

In sections of the medulla and pons numerous small gummata are to be seen lying in the meninges and in the periphery of the nervous substance. Some of the sections show very clearly that this gummatous infiltration is a direct extension of that in the meninges, and in general it may be stated that all the gummata have this connection.

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Besides the tumors noticed above there were others found here and there in the base of the brain.

Spinal Cord.—After having been hardened in Müller's fluid, the fresh cut surfaces of the spinal cord show that the right lateral column is less deeply colored than the other white tracts. The left lateral column is also of a somewhat lighter color than the surrounding tracts. At the commencement of the lumbar enlargement the anterior gray horns appear diseased; a little lower down hemorrhages are evident in them, and still lower the gray matter seems to have been disintegrated, leaving a cavity. Just below this there is an area in which the gray matter, although evidently diseased, presents no loss of substance. This is again followed by hemorrhages and cavity

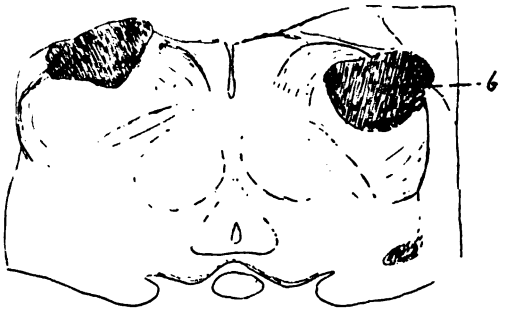

FIG. IX.

formations in the anterior horn. Below this second more pronouncedly diseased area, the gray matter seems to be normal.

Sections were cut from the different regions of the cord and stained in haematoxylin and eosin, carmine, and by Weigert's haematoxylin method.

The pia mater throughout the whole length of the cord is infiltrated with small round cells. This infiltration is most intense about the anterior longitudinal fissure, and is best seen in sections cut from the diseased area of the lumbar enlargement (Fig. x). The vessels which run in the pia mater are usually surrounded by an intense cellular infiltration, and some of the smaller ones are obliterated by it. The infiltration accompanies the vessels as they run into the fissures, and to some extent into the cord. The connective tissue surrounding the nerve roots is more or less affected.

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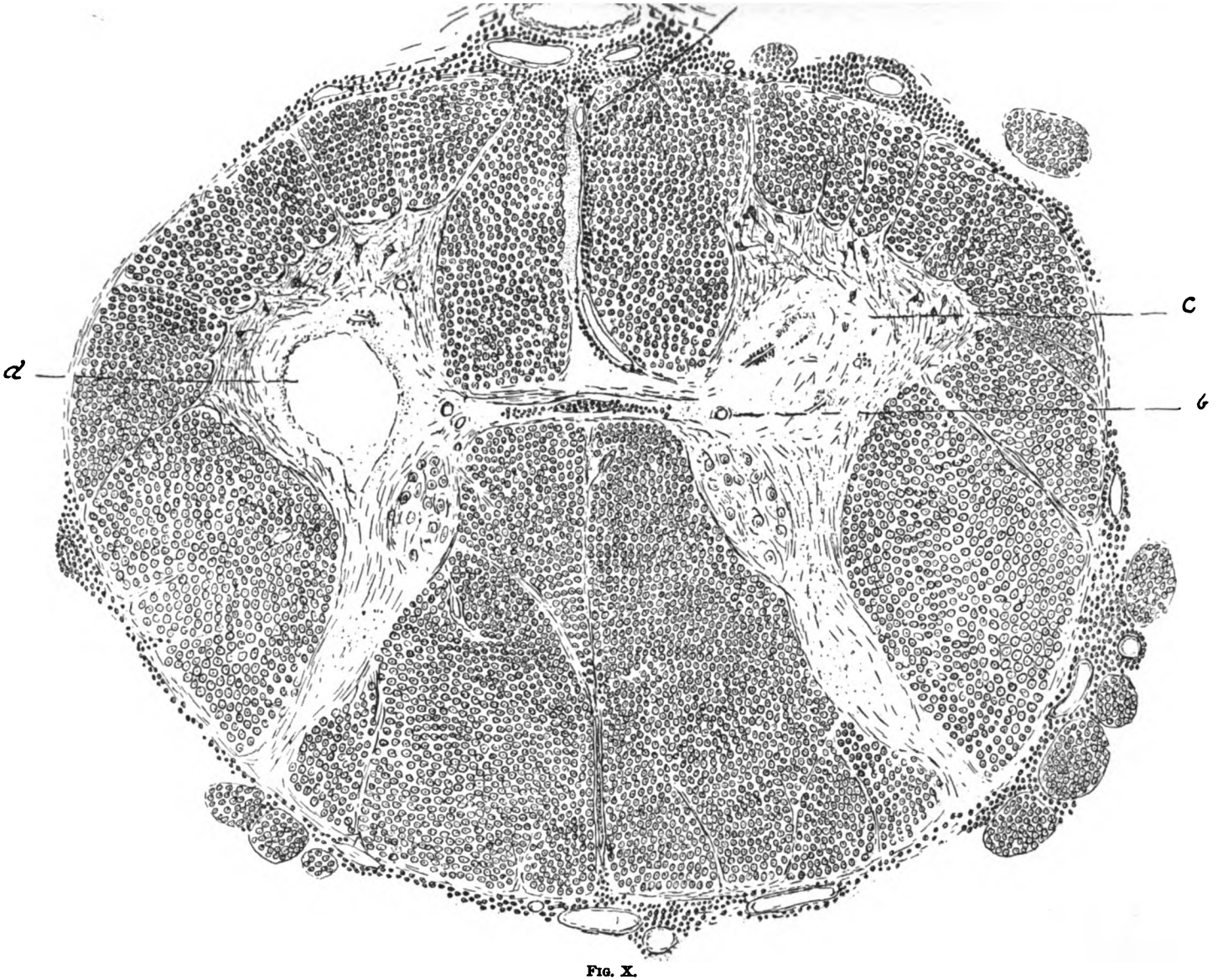

FIG. X.

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The walls of the larger vessels, which are seen in cross-section lying about the cord, seem to be but little affected, except by the cellular infiltration of their adventitia. The internal coat shows no marked thickening. Many of the smaller vessels present a very striking condition of their coats, these having undergone a hyaline degeneration. This is well seen in the vessels running in the anterior and posterior median fissures (Fig. X, *a* and XI) and in the vessels which are seen in cross-section at the root of the anterior horn (Fig. X, *b* and XII), and in the little vessels running in the gray matter.[1] The

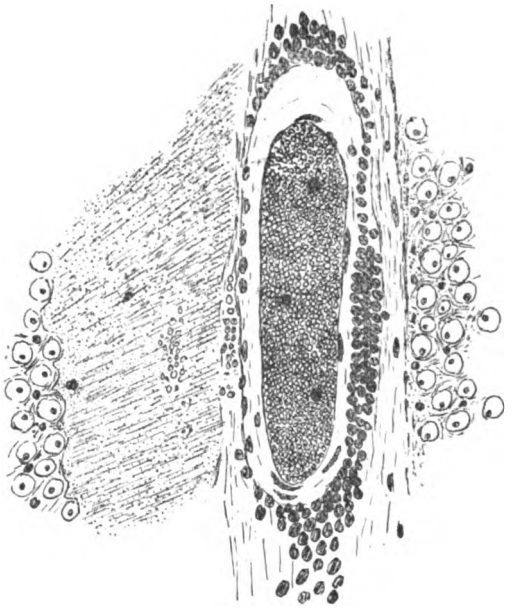

FIG. XI.

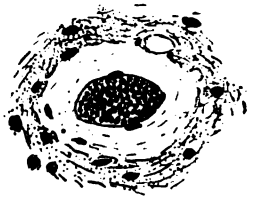

FIG. XII.

walls of the vessels which present this change appear homogeneous with but very few scattered nuclei, and are highly refractive. This hyaline substance, if such it be, does not stain to any extent in sections prepared as these were. The vessels are usually surrounded by small round cells. This change in the walls of the vessels is to be seen in sections from every portion of the cord; it is also present

[1] Figs. XI and XII are cross sections of Fig. X at *a* and *b* respectively, shown under a higher power.

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in sections of the medulla and pons, and some of the vessels running among the nerve roots in the cauda equina show this change. Some also in this situation show a typical endarteritis obliterans (Fig. XIII). It is, however, most marked in the lumbar region where the degeneration in the anterior horn is present.

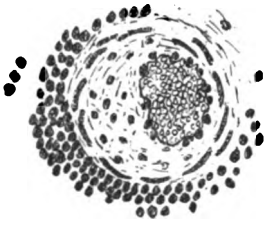

FIG. XIII.

The nerve roots as seen on sections from the different parts of the cord are for the most part normal, although the connective tissue surrounding and separating the nerve bundles is often infiltrated with small round cells. The blood vessels running in the nerve bundles are more prominent than is usual and are often surrounded by a cellular infiltration. Sections cut from the nerve roots, which

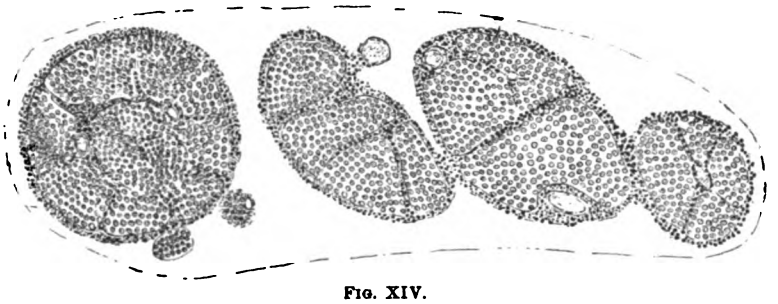

FIG. XIV.

run into the gumma on the third cervical nerve (Fig. XII), show that near the periphery of the tumor the cellular infiltration is confined to the connective tissue elements. The nerve fibres, just before they enter the tumor mass, seem remarkably well preserved. The nerve roots making up the cauda equina show no marked abnormality.

The gray matter in the cervical region is nearly normal. It appears somewhat more richly supplied with blood than is usual,

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and there are here and there small capillary hemorrhages. The large multipolar cells in the anterior horns are well preserved and are present in about normal numbers. In sections from this region, stained by Weigert's haematoxylin method, the lateral columns appear macroscopically less deeply stained than the other white tracts. Under the microscope it is difficult to convince oneself of any marked change in the nerve fibres of these columns. The axis cylinders are present in normal number, surrounded by thin myelin sheaths, which do, however, appear somewhat distended. The connective tissue is not noticeably increased.

The condition in the thoracic region of the cord is practically the same as that seen in the cervical.

The microscopical examination of the diseased portions of the lumbar region reveals here very interesting changes. Besides those changes described above as affecting the meninges and blood vessels, there is a most extensive destruction of the gray matter. In the anterior horns, at the points where the process is apparently commencing, the blood vessels are conspicuous, their walls usually having undergone the hyaline degeneration, and often infiltrated with small cells. Capillary hemorrhages are frequently seen surrounding these vessels. The neuroglia has lost its typical structure and has become rarified, staining here much less deeply than in the undiseased regions. It presents a finely fibrous appearance, and here and there branched neuroglia cells are seen. The smallest vessels are easily seen on account of their swollen hyaline walls. The large ganglion cells, which should occupy this affected area, have either completely disappeared or are represented by their shrunken and degenerated remains. These changes do not usually occupy the whole of the anterior horn, but are, more or less, limited to certain portions of it. The disease affects more especially the central part of the gray matter, leaving its superior and outer portions comparatively unaffected (Fig. x, *c*).

At the places where this destructive process has reached its greatest intensity, there is a complete loss of substance in the gray matter (Fig. x, *d*). These cavities, which occupy for the most part the central portion of the anterior horn, but which extend somewhat into the posterior horn, are surrounded by an area of degenerated tissue. This tissue is extensively infiltrated with well-preserved red blood

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corpuscles. In places near the border of the cavity, one sees the deposit of a homogeneous finely granular substance, which stains readily with eosine. This substance is seen usually in connection with blood-vessels, and is apparently the same as that which accompanies the vessels running in the anterior fissure (Fig. x, *a*).

In sections through the lower portion of the lumbar enlargement, the gray matter is normal, containing a great many large, well-stained ganglion cells and normal nerve fibres.

The white tracts in the lumbar enlargement present practically the same condition as that described in the thoracic region.

A short review of this case may help us to keep in mind what seem to be its important features:

The patient, a man thirty-three years old, acknowledged to having had gonorrhœa, but denied any syphilitic infection; he began to complain of intense headache about one year before being admitted into the Hospital. In January, 1889, he consulted Dr. Hiram Woods on account of double vision. He was at that time found to have paralysis of the right external rectus muscle; this paralysis was completely cured under the use of iodide of potassium. In May of the same year, he again visited Dr. Woods, who discovered a weakness of the superior oblique muscle of the left eye, the other muscles of both eyes acting normally. There were no opthalmoscopic changes in the fundus of either eye. Dr. Woods then lost sight of him, and the patient applied for admission to the Hospital on the 13th of November, 1889, two weeks before his death. He was then unable to give an account of himself, but his friends said that he had been suffering for about a year with very severe headaches, and with difficulty of vision; also that he had complained from time to time of some numbness on the right side of his face. He had stopped work a month before, and had been in bed three weeks. The examination showed that the patient had ptosis on the left side, the left pupil was dilated and fixed, the left eyeball could only be moved outwards; *i. e.* there was complete paralysis of the left third and fourth nerves. The right eye showed no abnormality. There was no optic neuritis. Sensation on the right side of face seemed somewhat duller than on left, and the muscles on the whole right side of the body were much weaker than those on the left side. The deep reflexes were everywhere exaggerated.

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Not knowing at that time the history which we subsequently got from Dr. Woods, and finding a paralysis of the left third and fourth cranial nerves, combined with a weakness of the muscles on the right side of the body, it seemed clear that there must be a growth on the left crus, and that such a lesion would explain all the symptoms.

The autopsy revealed a growth on the left crus, involving the third nerve; besides this, the expected tumor, there were many others whose presence had not been thought of; thus the left fourth nerve, the right fifth, sixth, ninth and twelfth nerves were all more or less involved in tumor masses. Other growths were found in the crura on both sides and in the under surface of the brain. Marked changes were also found in the spinal cord.

The endeavor to bring the symptoms noticed during life into any sort of accord with the condition found at the autopsy, seems at first sight nearly hopeless, and indeed it only can be done in a very imperfect manner. In the first place, it must be acknowledged that we noticed no symptoms to correspond to the very marked changes which were found in the spinal cord. It should be remembered that the condition of the patient when first seen was such as to make the examination difficult. What we did find was a partial right hemiplegia with slight sensory changes, combined with a paralysis of the left third and fourth nerves, and we assumed, a growth on the left crus involving these two nerves. The fourth nerve, instead of being involved in the same growth, was the seat of an independent tumor. The final symptoms would have been the same in either case, and the history which was then unknown was the only thing that might have led to the proper diagnosis. The tumors on the other cranial nerves were all on the right side, and tended, with one exception, that on the sixth, merely to intensify somewhat the hemiplegic symptoms, and it is not possible to determine even now just the extent to which the symptoms depended on the lesions of the nerves or on those in the crus; the anaesthesia, for example, of the right side of the face was so slight that the tumors of the left crus might well have accounted for it if no lesion had been found of the fifth nerve.

The tumor on the right sixth nerve should have caused paralysis of the external rectus muscle of the right eye, a symptom which certainly ought not to have been overlooked, and having tested the movements of the right eye shortly before death, and found them

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normal, we were at a loss to explain its presence. Soon after the autopsy we learned by chance that Dr. Woods had seen the patient, and through his kindness we were able to use his notes, which threw a great deal of much-needed light on the case. It will be remembered that Dr. Woods treated the patient in January, 1889, for a paralysis of the right external rectus muscle, and that under anti-syphilitic treatment this muscle regained completely its normal function, and that when Dr. Woods saw him in May, it was still acting well. So it seems fair to consider that this nerve was performing its function at the time of the death of the patient, and several months preceding it, in spite of the presence of the gumma, which was found on it at the autopsy. The microscopical examination of this nerve beyond the tumor shows that, although it could not be considered normal, still it contained a good many well-preserved nerve fibres, a very different condition from that found in the left third and fourth nerves which were paralyzed at the time of death. Sections of this gumma (Fig. v) correspond exactly to those of the gummata on the other cranial nerves. I was unable to trace any nerve fibres running through the gummatous tissue, and I do not understand how so many fibres escaped destruction in it. We know that the granulation tissue, which precedes the gummatous formation, grows primarily in the connective tissue surrounding the nerve fibres (Fig. XIV), and that a nerve may be quite extensively involved in the process and still retain its function (Siemerling).[1] It does, however, seem remarkable that such a tumor, situated as this one is, should not have produced a total destruction of the nerve. I have been unable to find a record of any similar case.

Any symptoms that may have been produced by the other tumors which involved the left crus were masked by those caused by the original gumma. It is interesting that so many separate lesions should have been so arranged that the resulting symptoms could all be accounted for by the supposition of one tumor. The importance of the previous history of this case is obvious.

Besides the gummata mentioned there were others in various parts of the brain; these, with the exception of those in the right crus, were so situated as not to produce any focal symptoms. The latter

[1] Arch. f. Psychiatrie, XXII, p. 191.

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apparently affected the pyramidal tracts on that side, and should have caused weakness on the left side of the body. The patient, however, had as much strength on that side as seemed compatible with his general condition, and the only symptom which can be referred to this lesion is the exaggeration of the deep reflexes.

From the standpoint of pathological anatomy, this case is also of interest. The arteries at the base of the brain present a very typical example of the gummatous arteritis described by Baumgarten[1] and the only thing which requires special mention is the very perfect way in which some of the sections illustrate the point insisted upon by Thoma,[2] that the new growth of connective tissue in the intima of an artery is compensatory to a diseased and weakened condition of its media. On these sections there is a more or less sharply localized degeneration of the muscular coat, and corresponding to this, both in situation and intensity, there is a new formation of connective tissues in the internal coat.

The gummata on the cranial nerves and in the substance of the brain are typical and require no minute description. It was noticed in the description of the microscopical appearances that there was quite a good sized artery attached to each of the tumors involving the cranial nerves, and the thought suggests itself that the point where a vessel crosses a nerve may be peculiarly favorable for the growth of the syphilitic tumor.

The lesions found in the spinal cord are especially interesting, partly on account of the comparatively few records of autopsies in cases of syphilis of the spinal cord, but particularly from the occurrence of the local destruction of the gray matter in the anterior horns, which makes this case nearly if not quite unique.

The changes which are produced by syphilis in the cord, and its membranes have in the course of the last few years been the subject of a number of investigations. Greiff[3] in 1882 collected thirteen cases from literature, and reported a case of his own. Since then Eisenlohr,[4] Jürgens,[5] Rumpf,[6] Meigs and de Schweinitz,[7] Gilbert and

[1] Virchow's Arch., Bds. 76 and 86.
[2] Virchow's Arch., Bds. 93, 95, 101, *et seq.*
[3] Arch. f. Psych., XII, 564.
[4] Neurol. Centralb., 1884, p. 73.
[5] Charité Annalen, 1885, 10, p. 729.
[6] Der Syphilitischen Erkrankungen des Nervensystems, 1887.
[7] Jour. of Nerv. and Ment. Diseases, 1887, p. 1.

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Lion,[1] Schmaus,[2] Oppenheim,[3] Siemerling,[4] have all written important articles.

From these it seems that syphilis causes in the spinal cord essentially the same lesions as it does in the brain, *i. e.*, a diffuse cellular infiltration or the formation of gummata in the meninges accompanied by marked changes in the vessels. The nervous tissue itself is affected secondarily, either by the extension of these processes along the connective tissue septa, running into the spinal cord and giving rise to sclerosis or to the production of gummata; or the changes in the vessels may, by disturbances in the blood-supply, cause destructive changes in the nervous elements, producing sometimes softening and sometimes sclerosis.

The cellular infiltration of the meninges is almost a constant lesion, and is considered by many observers the primary change. It is widespread, most intense about the longitudinal fissures and can often only be recognized by the aid of the microscope. There may or may not be the formation of gummata in the membranes, the walls of the vessels on the nerve roots or in the substance of the cord. There is some difference of opinion concerning the changes in the blood vessels, but the prevailing view is that the vessel walls are affected by the extension of the cellular infiltration in the meninges to their adventitia and muscular coats, and that the increase of connective tissue in the intima is secondary to this. It seems, however, more probable that the vessels in this situation, as well as those in the brain and elsewhere, may be directly affected by the syphilitic virus and become the seat of a primary arteritis. The process affects the veins as well as the arteries, causing in this case a phlebitis obliterans.

The walls of the smaller vessels and those running in the cord have several times been found swollen and hyaline.

The lesions which were found in the spinal cord of the case described above correspond, for the most part, closely to the descriptions of the authors referred to. There are, however, some changes which deserve especial notice. The condition of the lateral tracts is

[1] Arch. Gen. de Médicine, 1889, p. 402.
[2] Deutsch Arch. f. Klinishe Medicine, 1889, XLIV, p. 244.
[3] Zur Kenntniss der Syphilit. Erkrank. des Cent. Nervensystems, Berlin, 1890.
[4] Arch. f. Psych., XXII, pp. 191 and 257.

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a curious one. When the cord, hardened in Muller's fluid, is cut across and the surface examined, the lateral pyramidal tracts are seen to be less deeply colored than the surrounding tissue. If sections be cut and stained in Weigert's hæmatoxylin, these tracts are again easily distinguished by their lighter color, but if these sections are examined under the microscope, they present here surprisingly few morbid changes. The only abnormalities that can be distinctly made out are a swelling of the nerve fibres and a fainter staining of the myelin than is usual. This appears to be an early stage of secondary degeneration.

The hyaline degeneration of the walls of the small vessels is in this case a widespread and striking lesion. It is well seen in the smaller vessels and also in the smallest vessels which are visible in the gray matter, especially in those portions that are diseased. Similar changes were noticed in the cases of Meigs and de Schweinitz, Schmaus and Siemerling.

In a paper on syphilis of the lung, Councilman[1] described a hyaline degeneration in the capillary walls in the lungs and kidneys of two syphilitic cases. He considers this the primary change and thinks we can best understand its production by "the assumption of a soluble substance, which having been produced by an organism, exerts a special action on certain tissues of the body—namely, on the capillary walls." In spite of the fact that "hyaline degeneration" is a somewhat indefinite term and probably includes more than one process, it is very suggestive that in so many of the cases of syphilis of the spinal cord the walls of the smaller vessels should have been described as hyaline.

The gray matter throughout the cord was found to be richly supplied with blood-vessels, and here and there it was the seat of small capillary hemorrhages. In general, there were no very well-defined alterations in the ganglion cells. But in the upper part of the lumbar enlargement there was found very extensive destruction of the gray matter. Here there is what is essentially an anterior poliomyelitis, accompanied by capillary hemorrhages, which has gone so far in some places as to cause the breaking down of the tissues and the formation of cavities of quite considerable size. Some of the

[1] Johns Hopkins Hospital Bulletin, February-March, 1891.

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sections look as if the formation of the cavities had been preceded by comparatively large hemorrhages.

Lesions of the gray matter in syphilis of the spinal cord have been described by several authors, but for the most part they have consisted in rather indefinite changes of the large multipolar cells in the anterior horns and Schmaus' second case[2] is, as far as I know, the only one in which there was anything that could be called a true poliomyelitis. In this case there was found in the lumbar region of the cord a localized destruction of the anterior gray matter, the description of which shows the process to have been very similar to that observed in our case. Schmaus also found a hyaline degeneration of the smaller blood-vessels throughout the cord, but affecting especially those in the posterior columns. He considers the process in the gray matter to have been inflammatory, and entirely independent of the changes in the blood-vessels, only being connected with these by their common cause, syphilis.[1]

In our case we consider it more probable that the changes in the gray matter were secondary and dependant upon the lesions of the blood vessels. In the present state of our knowledge, the supposition that the syphilitic poison can cause a primary inflammation of the gray matter is, to say the least, bold. Again, we know that disturbances of the circulation act as the cause of many of the morbid changes in the central nervous system. This has long been known in connection with the brain, and is every day becoming more and more recognized as an important factor in the pathology of the spinal cord. The experiments of Ehrlich and Brieger, and of C. A. Herter[2] have shown that changes in the blood supply of the cord, if continued even for a short time, may produce permanent destruction of the nervous tissue. Nauwerck[3] reports a case of softening of the spinal cord in which he found well marked lesions of the blood vessels, and these he considered to be the primary change, and that the softening was dependant upon them. Klebs[4] has lately reported a most interesting case of Landry's paralysis, in which the microscopical exami-

[1] Deutsch. Arch. f. Klin. Med. XLIV, p, 257.
[2] Journal of Nerv. and Ment. Diseases, Vol. XIV, No. 4, April, 1889.
[3] Beitr. zur Path. Anat. Zeigler, Vol. II, p. 73, 1888.
[4] Deutsch. Med. Wochenschrift, 1891, No. 3, p. 81.

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nation of the cord showed a hyaline thrombosis of its central arteries. He thinks that the symptoms were caused by the disturbance in the circulation of the gray matter which this lesion produced.

Therefore, finding as we did extensive alterations in the walls of the blood-vessels and a lesion of the gray matter which might be accounted for by a disturbance in the circulation, the conclusion that the one depended on the other was not far to seek.

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JOHNS HOPKINS HOSPITAL REPORTS

REPORT IN PATHOLOGY, I

AMŒBIC DYSENTERY

By WILLIAM T. COUNCILMAN, M. D., AND HENRI A. LAFLEUR, M. D.

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AMŒBIC DYSENTERY.

By WILLIAM T. COUNCILMAN, M. D., AND HENRI A. LAFLEUR, M. D.

In this article we propose to consider a disease which is characterized by definite pathological lesions, and is separated not only by its distinctive pathological anatomy but also by its ætiology and clinical history from other affections of the intestines with which it has hitherto been classed under the general name of dysentery. The word "dysentery" was originally used only in a clinical sense to designate a form of intestinal flux accompanied by pain and tenesmus. It was afterwards found that when pain and tenesmus were present in an intestinal flux, the colon or rectum, or both, were the seat of disease; and gradually the word became used in an anatomical sense to express a pathological lesion. Under this common name have been embraced diseases with lesions which differ in their character, mode of production, situation and extent, fully as much as the various forms of pneumonia differ one from another.

Some attempt has been made by pathological anatomists to divide the lesions of dysentery into various groups solely on an anatomical basis, but with the ill-success which must follow any classification of disease in which ætiology plays no part. It is only after we know the ætiology of a disease that we are able to study intelligently its pathological anatomy by following up the series of changes which the tissue under the influence of the causative factor passes through

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in the production of the specific lesions. We want no better example of this than is afforded by tuberculosis. In the disease we are studying, the lesions are not confined in all cases to the intestines. In other tissues, in the liver and lungs, we find lesions which differ from the intestinal lesions in their appearance, but are due to the same cause and pathological processes. The lesions may differ, but the differences are to be explained by their situation, their extent and the nature of the tissue in which they are produced; the pathological process is the same in all. In our study of this disease, we have had the advantage that lesions produced by the amœba differ in mode of production and kind from those produced by bacteria. We believe that this is the first instance in which lesions produced by amœbæ in animal tissues have been carefully studied, and there may be errors both in observation and judgment, which further observation in the same field will correct.

Although earlier observers had discovered certain organisms belonging to the protozoa in the stools, the first account given of the presence of amœbæ in the intestinal contents comes from Lambl [1]* in 1859. He mentions finding minute amœbæ in the stools of children suffering with enteritis. These amœbæ were found along with other organisms, and no special importance was attached to their presence.

Cunningham [5] found numbers of amœbæ in the stools of cholera patients. They were found in 18 per cent. of the cases examined, and were of large size, containing numerous granules and sometimes showing vacuolation very distinctly. He stated that they multiply

* Davaine,[2] in 1853, described certain small actively motile organisms which he found in the stools, and to which he gave the name of *cercomonas hominis*. He described under this name a small oval body with an actively moving cilium at both ends. Lambert [3] subsequently described the same organism under the name of *cercomonas intestinalis*, a delicate oval body .018 to .021 mm. in its long diameter, which he found in great numbers in the jelly-like discharges of children suffering from diarrhœa. Malmsten [4] found in the discharges from the bowels of two patients an oval ciliated infusorial animalcule about 0.1 mm. in length, which he named the *paramœcium coli* and of which he has giving a drawing. The first patient was a man thirty-eight years old, who had long suffered from chronic diarrhœa: the second, a woman thirty-eight years old, affected with chronic entero-colitis of which she died. The infusoria were found in the stools of both cases in great numbers. In the last mentioned case the solitary glands in the lower part of the ileum were swollen, and in the colon there were small necrotic ulcers which apparently had their origin in the solitary follicles. The organisms were found only in the large intestine, and were most numerous in the mucus which coated the intestine between the ulcers.

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by gemmation, and made some attempts to cultivate them. Out of the body they do not retain their characteristic appearance for any length of time and may disappear in twenty-four hours. He did not ascribe any importance to their presence. Lösch [6] was the first author who gave an accurate description of a species of amœbæ which he found in the stools of a dysenteric patient, together with a careful clinical history and account of the autopsy. This work of Lösch's really is the pioneer work on the subject; the observations were carefully made, and his description of the organism, to which he gave the name of *amœba coli*, was so good that but little has since been added to it. He claimed for this organism that it was the cause of the disease, and attempted to prove this by injecting stools of the patient containing the amœbæ into the rectum of dogs. The case which Lösch studied was received in the clinic of Prof. Eichwald, in St. Petersburg, November 1873. The man came to St. Petersburg in the summer of 1871, and there became affected with an obstinate diarrhœa, which lasted for several months and so weakened him that he could not sit up in bed. He returned home improved, but was so weak that he could not serve in the army in 1872. He recovered and returned to St. Petersburg in 1873. In August, he was again affected with diarrhœa, accompanied by fever and general weakness. He entered the hospital shortly after, became much better after being two weeks under treatment, and went out. The recovery, however, was not permanent, and he returned at the end of October. On admission to the hospital he was much emaciated, and there was slight œdema of the lower extremities. He had then seven to nine fluid stools daily, which were passed with considerable pain. The stools were very offensive, thin, of a reddish-brown color, and contained a quantity of greyish-red or whitish masses of mucus and pus, which formed an abundant sediment. On microscopic examination, they contained the amœbæ in great numbers. At first, under treatment, he became somewhat better; but subsequently the stools became more sanguinolent and frequent, and contained increased numbers of amœbæ. The aggravation of the symptoms always coincided with an increase in the number of the amœbæ. No medication seemed to ameliorate the symptoms or lessen the numbers of amœbæ until it was found on experiment that a solution of quinine 1 to 5000 sufficed to kill the amœbæ after exposure for one minute. The

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amœbæ became motionless, assumed a round form, became coarsely granular, and had all the appearances of dead amœbæ. A clysma of 20 gms. to a pound of water (1 part quinine to 288 water) was injected into the rectum, and followed shortly by a large injection of warm water. The amœbæ vanished for several days and then returned in the stools. The patient gradually became so emaciated and weak that transfusion was done. In March 1874, there was a severe cough and an attack of pleurisy, during which time the amœbæ disappeared. In April, the stools became sanguinolent again, and the amœbæ reappeared. The patient died on April 12th. At the autopsy the mucous membrane in the upper part of the colon was hyperæmic, œdematous and covered with partially broken-down sloughs. Further on, the mucous membrane was thickened and irregular on the surface, and covered with ulcers, some of which were cicatrising. The thickening was both general and in the form of round elevations. On microscopic examination, the thickening was found to depend chiefly upon an inflammatory infiltration of the submucosa. The flat, round elevations were formed by cysts in the submucosa filled with gelatinous contents. The amœbæ found in the stools were oval, pear-shaped or irregular in form, five to eight times the size of a red-blood corpuscle. They were formed of an outer hyaline or faintly granular material enclosing a more granular mass. They were often so abundant that a single field of the microscope contained from sixty to seventy. They changed their form frequently with great rapidity, thrusting out blunt processes which were at times rapidly withdrawn; at others the granular interior slowly flowed into them and a change of place resulted. The nucleus was pale, of delicate contour and difficult to make out. It always contained a nucleolus of variable size. In the resting amœbæ the nucleus was always in the midst of the granular interior. Refractive vacuoles of various sizes, which underwent some change of form, were very numerous in the central mass. Foreign substances, such as bacteria, fragments of cells and red-blood corpuscles, were frequently seen in the amœbæ.

One to two ounces of the stools containing the amœbæ were injected into the rectums of four dogs. In one of the dogs an acute inflammation was excited by the injection of croton oil before the injection of the stools. This case and two of the others were negative.

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In one dog three injections were made on successive days. He was sick for two or three days, lost his appetite and then apparently recovered. On the eighth day after the last injection a small lump of mucus in the well-formed stool was examined and found to contain amœbæ. The dog was killed on the eighteenth day after the last injection. The mucous membrane of the rectum was reddened, swollen, and in three places there were superficial ulcers.

Cunningham,[7] in a further and more extensive article on the development of the protozoa present in the intestinal canal, says that he has found amœbæ in the intestinal canal of healthy subjects, as well as in those affected with cholera and other diseases. He thinks that the conditions which especially favor their presence are fluidity of the stools and an alkaline reaction. They were never so abundant as in certain cases of cholera. He also found them in the dung of cows and horses. Along with the amœbæ he frequently found bodies which he regarded as their spores. These were spherical or elliptical cells from .035 mm. to .092 mm. in diameter, characterized by the clear oily character of their contents. Sometimes these were connected with one another by a delicate intercellular basis, which he thinks represent the structure described by Hallier as the "sporogenic cyst." The fluid which seemed best adapted for the culture of the amœbæ was a strong filtered solution of fresh cow-dung.

In the cholera report of Koch[8] there is an interesting account of the autopsies which were made on five cases of dysentery in Egypt, in two of which abscess of the liver was also found. In the colon of most of the cases there were œdema and extensive ulceration of the mucous membrane. On microscopic examination of the ulcers, amœbæ were found in the tissue at the base of the ulcers and in the material covering the ulcers, in all but one case, and in that the ulcers were entirely or almost cicatrised. The amœbæ were about twice the diameter of a leucocyte, of very variable form, with clear vacuoles and a number of granules or rods, which often resembled short, thick bacilli. The amœbæ were only found in the sections which were made through the base of the ulcers and stained with aniline colors. They were never found in the bloody mucus of the dejections, nor in the intestinal contents. In one of the cases of liver abscess they were found in the capillaries of the liver tissue adjoining the abscess; but in the wall of the abscess only masses of micrococci were found.

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In the other case neither amœbæ nor bacteria were found either in the wall of the abscess or in the surrounding liver tissue. From finding them always in the base of the ulcers, Koch thought it probable that they stood in close relation to the cause of the disease. Amœbæ have been found in the intestinal contents by four Italian observers: Grassi,[9] Perroncito,[10] Calandruccio,[11] and Blanchard.[12] The reports have reference only to their presence. Grassi saw them in small numbers in six cases of intestinal inflammation, but does not ascribe any importance to them. Leukart[13] says that his friend, Dr. Sousino, in Cairo, told him that he had found large numbers of amœbæ in the intestinal mucus of a child who died of dysentery.

Next to the paper of Lösch, the most important is a series of publications by Kartulis,[14–18] in Alexandria, Egypt, on dysentery and abscess of the liver. In his first publication[14] he gives a description of the amœbæ which does not materially differ from that given by Lösch, except in reference to their size. The dimensions given by Kartulis for the resting amœbæ vary from 0.012–0.030 mm. in diameter, the diameter given by Lösch being 0.02–0.035 mm. The plasma of the amœba he considers to be a mucoid fluid material, which he proposes to call "myxo-plasma," in contrast to the "hyaloplasma" of other varieties of amœba. To the inner mass containing numerous small refractive granules he gives the name "hyaloplasma." The nucleus is homogeneous, slightly refractive and ordinarily seated in the centre. Vacuoles are present in varying numbers, and sometimes granular material may be seen in these. At first, some difficulty was experienced in finding the amœbæ in the intestinal ulcers; but when the freshest, smallest ulcers were examined, they were present in considerable numbers. The stains found most satisfactory in demonstrating them were methylene blue and gentian violet, the tissue being hardened in alcohol.

The second paper in Virchow's Archiv[15] is devoted to the study of the relation of the amœbæ to abscess of the liver. In this he gives the results of inoculation experiments on monkeys, dogs, guinea-pigs and rabbits. The stools containing the amœbæ were injected into the rectum, but with negative results. He also attempted to cultivate the amœbæ in a culture medium of alkaline sterilised rabbit-dung, but was unable at this time to obtain a pure culture. He produced diarrhœa in two dogs by injecting these impure cultures, and amœbæ

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were found in the whitish diarrhœal stools, but the animals soon recovered. The dysenteric abscesses of the liver, he considers, are produced by the amœbæ which are carried to the liver by the portal veins. Of themselves, the amœbæ can only produce a certain amount of injury by tearing the capillaries, the agents which produce the abscesses being the bacteria which are carried within the bodies of the amœbæ from the intestinal canal to the liver.

In another article[17] he calls attention to the further distribution of the amœbæ, and cites cases in which they were found in Greece. In his last article on the subject[18] he gives the details of his experiments in the cultivation of the amœbæ, and bespeaks for the amœba as the cause of dysentery, a greater recognition among pathologists than it has hitherto had. His earlier attempts at cultivating them, in fluid blood serum and fluid gelatine, were not successful. The cultures in sterilised rabbit- and pigeon-dung were more successful; but he was never able in this way to obtain pure cultures. The culture medium which proved most suitable was an alkaline straw infusion at a temperature of 36° to 38° C. To this culture medium were added either contents of the liver abscess or stools containing the amœbæ. In twenty-four to forty-eight hours a thin membrane formed on the surface which consisted of bacteria and young amœbæ. Studied in drop-cultures, the amœbæ were much smaller than those in the stools; their movements in the swarm form were very active, but they did not send out pseudopodia. The nucleus and vacuoles were very clear after staining with aniline colors. Along with the amœbæ were small bodies which he regarded as free nuclei. The small amœbæ gradually become larger, send out pseudopodia and resemble those found in the stools. Among the actively moving amœbæ there were round inactive bodies with a nucleus and finely granular protoplasm; they were about the size of a white blood corpuscle; and the author considers them to be spores. (These last bodies described by Kartulis are possibly the same structures seen by Cunningham,[7] which were also considered by him as spores of the amœbæ.) Attempts were made to obtain pure cultures by successive inoculations, but this was only successful in one case in which the material for the first culture was obtained from a liver abscess in which no bacteria were found. With both the impure and the pure cultures inoculations were made on cats with positive results by

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injecting the cultures into the rectum and then tying the anus in order to have the material retained. The mucous membrane of the colon was found lax and swollen, with here and there small erosions. No regular ulcers were found. The author thinks that the ulcers are caused by the entry of amœbæ into the tissue, which they reach by passing between the glands of the mucous membrane. Feeding the animals with the amœbæ gave negative results.

Hlava[19] investigated the dysentery prevalent in Prague, and found the amœbæ in sixty cases, of partly endemic, partly sporadic dysentery. His description of them agrees fully with that of Kartulis. Hlava also injected the stools containing amœbæ into the rectums of dogs and cats with positive results in two dogs out of seventeen, and in four cats out of six. Kartulis[18] remarks that dogs become spontaneously affected with dysentery in Egypt, and the same amœbæ are found in their stools as in cases of dysentery in man. Hlava had negative results in rabbits, chickens and guinea-pigs.

Masiutin[20] found the amœbæ in five cases of intestinal trouble in the clinic of Prof. Lösch, in Kiev. Only one of these was a case of chronic dysentery. The others were: chronic intestinal catarrh, 2; typhoid fever, 1; acute intestinal catarrh, 1. From these observations the author does not believe that the amœbæ produce dysentery. He thinks that the amœbæ enter the intestinal canal in the drinking water, and increase there if they meet with suitable conditions. These favorable conditions consist in fluidity of the contents of the large intestine and increased secretion of mucus. He doubts if the amœbæ can of themselves produce ulceration, but thinks that the ulcers may favor their development in that they find a suitable soil for growth in the base of the ulcer.

Pfeifer,[29] in 1887, saw amœbæ similar to those described by Lösch in the stools of dysenteric children.

Osler[21-22] was the first to find amœbæ in America. He found them in the contents of a liver abscess operated on by Dr. Tiffany of Baltimore. They were present in large numbers in the creamy pus of the abscess, and were subsequently found in the stools of the same individual. The patient, a physician, had lived several years in Panama, and had there contracted a chronic dysentery with a subsequent development of liver abscess, which terminated fatally. The amœbæ described by Osler conform in all respects to the description

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of Kartulis, save in being a little larger. Lafleur[23] exhibited the amœbæ at a meeting of the Johns Hopkins Hospital Medical Society. They were from a case of dysentery in a sailor sailing between Baltimore and Bremen. He subsequently found the amœbæ in the sputum in cases in which an abscess of the liver had penetrated into the lung. A case was reported to the same society by Dr. Simon,[24] in which the diagnosis of the condition was made from the presence of amœbæ in the sputum.

Musser,[25] of Philadelphia, found amœbæ in the stools in three cases of dysentery.

Stengel[26] reports three cases of dysentery in which amœbæ were found in the stools.

Recently Dock[27] has reported twelve cases of dysentery in Galveston, Texas, in all of which amœbæ were found in the stools. In four of these cases abscess of the liver was also present. No microscopic examination of the tissues was made in the fatal cases.

The latest publication concerning amœbæ in the stools is by Cahen,[28] from Escherich's clinic in Graz. He found in the dysenteric discharges of a child four years old who recovered, amœbæ identical with those described by Kartulis, and differing from those of Lösch only in attaining a size two to three times the diameter of a red blood corpuscle, whereas Lösch assigns a diameter exceeding five to eight times that of a red blood corpuscle. Cahen recognized a nucleus in the unstained motile amœbæ, but could detect none in amœbæ stained with methylene blue. Attempts at cultivation and at inoculation of cats were unsuccessful. He appears to have found similar amœbæ also in all cases of dysentery which he examined for this purpose and to have missed them in other intestinal affections, but he gives no further details as to these observations.

There have been but few critical notices in recent medical literature on the relation of these amœbæ to dysentery. Baumgarten,[30] speaking of the opinion first positively expressed by Kartulis that the amœbæ are the cause of dysentery, expresses himself as follows: "We will not contradict this view, although recent observations and some of former date have shown that similar forms of amœbæ are found in the fæces in other intestinal affections, and even in the fæces of healthy individuals. We regard it as improbable that the amœbæ can produce the entire series of changes which constitute the dysen-

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teric process. Anatomically, dysentery consists in a combination of diphtheritic and purulent inflammation, which rapidly produces deep ulcerations of the parts affected. There is no analogy to make us believe that amœbæ can be the cause of the ulceration. We would think it more probable that the pyogenic micro-organisms play an important part along with the amœbæ in the production of the tropical dysentery." The recent observation of Kartulis, who has found in the abscesses of the liver colonies of pyogenic bacteria, along with the amœbæ, seems to speak for this view. How the amœbæ act in conjunction with the bacteria in the production of these lesions must be left for further investigation.

Baelz,[31] in Tokio, has recently reported a very interesting case in which amœbæ were found in the bladder. The case was that of a woman twenty-three years old, with tuberculosis of the genitalia and the lungs, who complained of great pain in the bladder. The urine was bloody; it contained pus cells, particles of necrotic tissue and great numbers of amœbæ. These were somewhat larger than those described by Lösch, but in every other respect were similar. The same forms were found in the vagina.

In reviewing the literature which has appeared on this subject up to the present time, we find that the presence of amœbæ in the stools of dysenteric patients and in the abscesses of the liver which follow dysentery has been confirmed by a number of observers. These observations have been made in the so-called tropical dysentery of Egypt; and in the endemic and sporadic dysentery of Europe and America. It is greatly to be regretted that, with the exception of Cunningham's reports, there have been no observations on the subject in India, which may almost be regarded as the home of the disease. Nor have there been any reports on the subject from French observers in Algiers. Kelsch and Kiener[32] in the article on dysentery in their work on diseases of the tropics do not mention the amœbæ. Some observers have also seen them in other intestinal diseases than dysentery, and even in the fæces of healthy individuals. With the exception of the investigations of Kartulis on liver abscess, there have been no observations published on the pathological anatomy of the disease. Our object in this paper is to study the diseased conditions in which the amœbæ are present, both from a clinical and from an anatomical point of view, to determine whether the amœbæ are always associated

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with definite anatomical lesions which can be separated as a unit from a number of different anatomical conditions which have been described under the term "dysentery," and whether there are distinctive clinical symptoms which accompany the anatomical lesions or not.

With our present knowledge of the protozoa we have not the same means of classifying and recognising species of amœbæ that enable us to recognise the various bacilli. Until we have such means it is impossible to say whether or not the amœbæ which have been found in the intestinal contents under varying conditions, are all one and the same species. It is probable that in the future we must recognise a number of different organisms as the cause of the various sorts of dysentery; for it is impossible that conditions so varying, in both their anatomical and clinical aspects, as are the various forms of dysentery, can be due to a single organism or even a single group of organisms.

We have called the organism, which was first described by Lösch under the name of amœba coli, the "amœba dysenteriæ." The name given to it by Lösch is not distinctive; for it seems very probable that there may be a number of species of amœbæ, which under certain conditions and in certain localities may inhabit the colon. This is evident from the work of Cunningham, who found them not only in the human fæces, in health and in disease, but also in the fæces of some of the domestic animals.

In our investigations made on cases in the Johns Hopkins Hospital and elsewhere in Baltimore, we have not found the amœbæ to differ materially from the description which has been given of them by Lösch, Kartulis and Osler. In searching for the amœbæ in the stools, there is some choice in the portions of the stools to be examined. They are rather more numerous in the small gelatinous masses which are often contained in the fæces, than elsewhere. The numbers of them which are found in the different cases and even in the same case, at different times, vary greatly. At times no single portion of the fæces which can be examined is free from them. We have frequently found them in such numbers that the entire field of a Zeiss D was filled with them. In other cases single examples of them were found only after long and careful search. Their numbers in general are proportional to the severity of the lesions. They were most abundant in the cases in which the lesions were most acute and most extensive.

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The amœbæ differ somewhat in appearance according as they are active or inactive. In an inactive condition they are round or slightly oblong, and are more refractive than the other cells which may be found in the fæces. In this resting condition there can frequently be distinguished no division into an ecto- and an endosarc, but there is simply a body, enclosing vacuoles of greater or less size. The vacuoles are clear and their contents are much paler than the substance which encloses them. They vary greatly in size. Some are found one-third the diameter of the entire amœba; others are much smaller, and in some cases the whole mass is so filled with tiny vacuoles of equal size, that the amœbæ resemble a compound granular body. In other cases there is a sharp division between the outer and the inner portions of the organism. The outer portion is composed of a pale hyaline or homogeneous substance, and the inner contains vacuoles, and is much more refractive. They are difficult to recognize in this condition unless active ones are found at the same time, and in this way the attention be directed to their presence; otherwise they may be overlooked. Again the swollen connective tissue cells in the fæces and the compound granular bodies may be mistaken for them. Their appearance when moving is characteristic. The activity of their motion varies greatly in the different cases, and even in different observations made on the same case. Every degree of activity of movement can be seen. At times the movements are so slow that one must look carefully for some time to be sure that any motion is taking place, and at others the movements are so rapid that certain of their phases can scarcely be followed. The movements consist essentially of two kinds; a progressive movement, and one limited to the thrusting out and retraction of pseudopodia. In some instances, there seems to be a certain rhythm in these movements, which occur at regular intervals. An amœba in which no movement can be detected will suddenly make one or several quick movements, thrusting out large pseudopodia or even changing its position, then become perfectly quiescent, suddenly repeating the movements after an interval.

In many of these sudden movements there is simply a change of form and protrusion of pseudopodia. The pseudopodia are always blunt. Several pseudopodia are frequently thrust out at the same time, the amœbæ then presenting a very irregular contour. In this stage of activity there is usually a division into ectosarc and endosarc;

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but in some very actively moving amœbæ, on a warm stage, the distinction between ectosarc and endosarc was entirely absent, the whole amœba flowing along simultaneously as one mass of protoplasm, vacuolated and containing foreign bodies.

The pseudopodia are of the same hyaline homogeneous mass as the ectosarc. We have seen one instance in which the entire amœba was filled with very large vacuoles, and there was no differentiation into the two structures; and then when the pseudopodia were thrust out, some of the vacuoles diminished in size as though their contents had gone into the protrusions. Some of the pseudopodia rapidly increase in size; the inner contents flow into them, and the amœba changes its position. Sometimes there is a continual progression in this way for some time in a definite direction. At others, there is an altogether different motion—a steady crawling or rolling motion, the ectosarc pushing out continually in one direction followed by the contents. In other cases, a very peculiar motion was seen; very large and long pseudopodia were thrust out, the extremities of which became connected with the amœba at some other point, and in this way an elbow was formed which quickly disappeared. Some of these pseudopodia were so long that they completely encircled the amœba, becoming again connected with it near the point of exit.

Various bodies were frequently enclosed in the amœbæ. The most common of these were red blood corpuscles, which were often found in considerable numbers, as many as six or eight having been seen in a single amœba. Many of these were perfectly preserved and could be easily recognized; others had lost their color and could be recognized only from their outline. Pus cells, well preserved or in various stages of disorganisation, were also seen, either in the ectosarc or in the vacuoles. Both bacilli and micrococci, and the spores of the former were sometimes seen in them. Not only were red blood corpuscles contained in the amœbæ, but blood pigment as well, under two forms. There were not only irregular brown masses of pigment, but in some of the amœbæ there were distinct black granules, similar to those found in the malarial parasites. The granules were often arranged in the form of a distinct ring. This was more often seen in the amœbæ in the sections than in the fresh forms.

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The endosarc is not granular: it seems to be composed of a dense homogeneous substance which is very refractive. The appearance of granulation is due to the very small vacuoles which it encloses. When the vacuoles are very numerous the endosarc has the appearance of a refractive net work. In most cases it has a distinctly greenish shimmer. Nothing corresponding to the contractile vesicle of the ordinary fresh-water amœbæ was seen. Many of the vacuoles changed their size, but there were none which underwent rhythmic contractions; nor was there anything which corresponded to the refractive vesicle. Contrary to the observations of those who have preceded us, we have found that the nucleus cannot definitely be made out when the amœba is examined in the fresh state. The difficulty seems to be in distinguishing it from the great number of vacuoles. Nor could any other details of structure other than the division into the ecto- and endosarc and the vacuolation be made out. This is the more remarkable because in the examinations which we made after hardening the amœbæ in the tissues, there was in some cases a very definite and peculiar structure.

The movements are very much accelerated when the examinations are made on the warm stage. We have found most convenient for this purpose the warming apparatus of Zeiss, in which the entire microscope rests in a box enclosed on four sides by warm water. When the movements have entirely ceased, they can be started into activity again by heating the slide. They may continue for a long time at ordinary room-temperature. In one case movements were seen after ten hours. On a cooled slide or on a slide to which some carmine solution had been added, a peculiar circular movement of the ectosarc was observed. The amœba protruded a broad pseudopodium to a short distance, and this, more or less rapidly encircled the body. This was repeated several times, sometimes in rapid succession, and always in the same direction, giving the appearance of protoplasmic waves. Gradually this movement became less active and frequent, until the amœba became perfectly quiescent and assumed a circular outline. Movements of this sort seemed frequently to precede the stage of quiescence. Sulphate of quinine solution, 1–5000, when placed at the edge of the cover-slip produced peculiar changes in actively moving amœbæ. As the solution diffused itself under the cover-slip, the active movements of the amœbæ were suddenly

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arrested, and they immediately assumed a circular outline becoming pale and blurred.

The amœbæ were further studied in various ways. The fluid containing them was spread on glass-covers and allowed to dry. A large number of these preparations were made from the last autopsy. Portions of fæces containing them in large numbers were placed in Müller's fluid, and sections made after embedding the mass in celloidin. The fluid dried on the slide was stained in various media, both the blood-stains and the bacteria-stains being used. None of these methods gave such satisfactory results as were obtained from the study of the amœbæ in the tissues after hardening in various media. Of all these media, the best results were obtained by hardening very small pieces in Flemming's solution, and then staining deeply in safranin. Good results were also obtained in the sections which were hardened in alcohol and then stained with methylene blue.

Each of these methods of hardening and staining the amœbæ gave results which differed in certain particulars. In sections of tissues hardened in alcohol and stained with methylene blue, the amœbæ are probably more easily recognised than in tissues treated by any other method. They are not stained quite so intensely as the nuclei of the cells. With a low-power they are easily recognised as dark-blue objects, several times the size of the surrounding cell-nuclei. Even with the low-power they often present quite a peculiar appearance, one portion of the body being intensely stained, the stain gradually fading off. This frequently gives them a distinctly crescentic appearance, which is not so apparent under a high-power. With a high-power, a stained and an unstained portion can be distinguished, the whole surrounded by a distinct cell-wall. The stained portion contains vacuoles which are often fully as distinct as in the fresh specimen. The staining is never perfectly homogeneous. In all cases the body appears, more or less, granular, and this is in some instances so marked, as in Figs. 1 and 2, Plate VII, that granules much brighter stained than the rest are seen, both in the stained and the unstained portions. The portion stained corresponds to the endosarc. These granules are round, and might be mistaken for micrococci; but they vary in size and in the intensity with which they stain. These granular amœbæ were most frequently found in the intestinal ulcers. In No. 2, of this series, an included red blood corpuscle "*a*" is seen.

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The nucleus does not stain with methylene blue, and cannot be distinguished in the specimens which are stained with this alone. Hæmatoxylin stains the specimens hardened in alcohol much like methylene blue. The amœbæ have very much the same appearance with this stain, but the granulation is not so evident. The small granules so intensely stained with the blue are only faintly, or not at all, stained with the hæmatoxylin. The nucleus does not stain in hæmatoxylin. In the same specimens stained with picrocarmine, the amœbæ are rather more brightly stained with picric acid than the tissues, but not so brightly as the muscular tissue and the blood.

In some respects the most perfect amœbæ were found in tissues which had been hardened in the heat for two days in Müller's fluid, which was accidently made much stronger with bichromate of potash, than the formula calls for. In some of these, as in Figs. 4, 5 and 6, Plate VII, the division into the central endosarc and the outer ectosarc is apparent; in Figs. 7 and 8, Plate VII, there is none. The large clear space in Fig. 5, Plate VII, has rather the appearance of having resulted from the rupture into one another of a number of large vacuoles. All of these specimens were stained with eosin and hæmatoxylin; and they have much more affinity for the eosin than for the hæmatoxylin. The nucleus is evident in most of these. It is a round or sometimes slightly irregular body of about the size of a red blood corpuscle. It has a distinct sharp contour and something of the vesicular appearance of an epithelial nucleus. In these specimens there is frequently a refractive body in the middle of the nucleus. The nucleus is distinctly disc-shaped and lies close to the surface.

In specimens showing the ecto- and endosarc, the nucleus always lies at the edge of the latter, and, when seen in profile, has not more than the thickness of a red corpuscle. It stains with eosin and more sharply with acid fuchsin. In some of these specimens stained with the latter, the periphery of the nucleus is stained so sharply that it appears as a bright ring, not unlike some of the forms of the malarial parasite in the red corpuscle. The nucleus is much more evident, however, in specimens which had been hardened in Flemming and stained with safranin. These specimens are especially interesting because certain details of structure in the amœbæ were seen which have not been described up to the present (that is to say, we have not found any descriptions of them). In general, the amœbæ appear

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larger in these specimens than after any other method of hardening. See 9, 10, 11, 12, and 13, Plate VII. The contents in many cases appear to have shrunken away from the cell-wall, leaving a perfectly clear space all around this, or the central mass is connected with it by slight and irregular prolongations. The central mass is faintly stained, has a somewhat granular appearance, and the vacuoles can be made out in it, but they are not so distinct as in other specimens. A nucleus can be seen in almost every individual; and in some it is stained much more brightly than in others. Its discoid shape and its position in the body are very evident. It frequently contains a central nucleolus; and in some instances, as in Fig. 9, Plate VII, brightly stained processes seem to be attached to the rim of the nucleus. Even in specimens hardened in Flemming, which are entirely unstained, the clear vesicular nucleus is easily distinguished.

In a few of the amœbæ from the abscess in the liver and lung there were intensely stained short rod-like bodies, generally curved and greatly resembling tubercle bacilli stained in fuchsin. Plate VII, Figs. 11 and 12. These were either single, in pairs, or sometimes three or more in a group parallel to each other. We could not make out the nature of these bodies until we saw similar bodies lying in the surrounding tissue. We regard them as nuclear detritus, either resulting from the digestion of cells by the amœba or taken into the amœba from the surrounding tissue. In Figs. 10 and 13, Plate VII*a*, we see nuclei of cells which the amœba has taken up. In 10, along with this, there are four red blood corpuscles.

The peculiar structures to which we alluded are seen in Figs. 13 and 14, Plate VII*c*. Both of these specimens are from an abscess of the lung. In these amœbæ there are aggregations of rod-shaped structures more brightly stained than the remainder of the tissue. They have a distinctly radiate arrangement; and in some instances, the nucleus seemed to be the point from which they radiate towards the periphery. The rods are very small and difficult to analyse; but under the Zeiss apo. 12, it can be clearly shown that they are rod-shaped structures. They were never seen in the amœbæ in the liver abscesses or in the intestinal ulcers. They were found in one of the amœbæ in a dried specimen which had been stained in methylene-blue. It is hardly possible to regard these structures as material which the amœba has taken up from without, for the distinct radiation of the

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entire mass and the equal diameter of the individual rods which compose the mass speak against this view. It may be that it is a structure of the organism which is only apparent under certain circumstances.

These specimens stained in Flemming show another thing, viz.: that the amœbæ rarely take up fat. On the edge of the lung abscess and in the alveoli, surrounded by fatty degenerated cells and tissue, they never contain any fat. In one or two instances only brownish granules were found in them, which might have been fat. There were none of the characteristic round black masses which were in all the surrounding cells in the tissue or free.

Sometimes along with the large and characteristic amœbæ, much smaller bodies, one-half the size of the usual forms or even less, are found. In none of these was a nucleus found. Generally they represent simply a more or loss granular mass, as in Fig. 7; but sometimes, as in Fig. 15, they contain numbers of vacuoles.

Specimens which are hardened in chromic acid show the amœbæ very much as do those hardened in Flemming. The nuclei are rather better stained in this than they are in Flemming; but the other details of structure are not made out.

The only reference we have found that might in any way refer to the radiate bodies, represented in Figs. 12 and 13, is in a statement by Bütschli.[98] He says that the protoplasm of *Pelomyxa* often contains peculiar small rod-like bodies, which surround the refractive body. These rods appear hyaline and may be of 0.008 μ. in length. No details of structure have been seen in them. The figure which he gives of this shows the rods forming a regular structure around the refractive body.

There was nothing of special importance brought out in the specimens which were stained on cover-slips. The amœbæ in a great measure lost their characteristic vacuolation in the drying. The nucleus appeared as in the hardened specimens and stained in aurantia like the blood corpuscles. Fragments of cells contained in them stained blue or green.

The clinical study is based on an analysis of fourteen cases of amœbic dysentery and extends from February, 1890, to July, 1891. In the general clinical review a comparison is made between this form of dysentery and cases of diphtheritic dysentery and catarrhal dysentery, in which the course of the disease presented marked differences

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from that observed in our cases of dysentery, and the absence of amœbæ in the evacuations was constant.

Of the cases of amœbic dysentery one was of tropical and one of doubtful origin; the remaining twelve were endemic.

CASE I.—*Dysentery, chronic, with exacerbations. Abscess of the liver. Operation. Amœbæ in the abscess pus and in the stools. Death.* Reported by Osler.

Dr. B., aged 29, resident in Panama for nearly six years, where he had suffered from chronic dysentery, came to Baltimore in May, 1889, and shortly after left for Germany. He had intervals of freedom from the diarrhœa, but in Vienna it recurred severely. Shortly after his return to America in December, 1889, he had an irregular fever, occasional chilly sensations and sweating, with progressive emaciation, and he became very sallow. On February 15th, 1890, it was observed that his liver was slightly enlarged anteriorly but not sensitive to pressure, while posteriorly there was a marked extension of the upper limit of liver dulness. He had from six to eight stools daily containing mucus and a little blood. At the end of another month, notwithstanding a diminution of the diarrhœa and an improvement in his appetite, he had lost flesh and strength. Anteriorly the liver dulness was not much increased; but posteriorly it extended a hand's breadth above the normal limit. There was tenderness on deep pressure below the right costal margin, and the patient complained of a dragging pain about the liver when lying on his left side.

On March 22d, after a preliminary exploratory aspiration, two large abscess cavities in the right lobe of the liver were opened and drained. The pus obtained was thick, of the color and consistency of cream, and in places slightly bile-stained. It contained actively moving amœbæ in large numbers. On each succeeding day at the time of dressing pus was removed from the drainage tube before irrigation was begun. On the first two days the amœbæ were quite numerous and very active, but on the three following days moving forms were less numerous, probably owing to the fact that stronger solutions of bichloride of mercury were used for irrigation of the abscess cavity. They subsequently became very numerous and were found daily until the death of the patient. After the

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operation the dysenteric symptoms did not abate, and there were from eight to sixteen movements daily, of very varied appearance. Some were entirely mucoid with a small amount of purulent and greyish shreddy material. Others were greenish and pultaceous like spinach, with occasional large necrotic-looking masses. Still again, others were composed of a large amount of dark brownish liquid in which floated grayish-white masses the size of a large pinhead often embedded in blood-stained mucus. Actively moving amœbæ were found daily in these stools and were particularly abundant in the small greyish-white purulent masses described above. The patient sank gradually and died of exhaustion on April 5th, 1890. There was no post-mortem examination. At the end of his report Osler observes that "it is impossible to speak as yet with any certainty as to the relation of these organisms to the disease. The subject is deserving of extended study, and a point of special interest will be the determination of their presence in the endemic dysentery of this country."

CASE II.—*Dysentery. Consolidation at the base of the right lung. Cough with bloody purulent expectoration containing elastic tissue fibres. Obscure physical signs pointing to deep-seated excavation in the lower lobe of the right lung. Amœbæ found first in the sputa, subsequently in the stools. Exploratory aspiration of the liver and lung with negative result. Operation. Abscess involving liver and lower lobe of right lung. Death from exhaustion. Autopsy.*

Wm. K., aged 48, was admitted to the Johns Hopkins Hospital on April 30th, 1890. He was born in Germany, but had lived in America for over twenty-five years and had never been south of Baltimore, where he was engaged at the harbor as a stevedore. He had never been ill until three weeks before his admission to the Hospital, when he had diarrhœa with severe cramps in the abdomen, especially at stool. He had a little fever, his abdomen was swollen and tender, and he passed five or six liquid stools daily. He kept at work and the diarrhœa increased to ten or twelve thin watery movements containing slimy material of a greenish color but no blood. On April 27th, after a purgative, the pain and looseness diminished, but returned on the following day. At the time of his admission he

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complained of fever, headache, slight cough and shortness of breath, and diarrhœa. He had not been confined to his bed though he was very weak and could eat little of anything. Vomiting had occurred only once in the course of his illness.

Physical examination.—Powerfully built and well nourished, face flushed, skin moist, tongue covered with a white pultaceous fur. Temp. 103.6° F., max. 104.8° F. at 8 p. m. Pulse 104, regular; resp. 24. Chest expansion equal on the two sides. Examination of the lungs: negative. A soft blowing systolic murmur over the præcordial area, but no increase in the area of cardiac dulness. Abdomen soft and relaxed; sensitiveness to pressure elicited along the line of the colon, particularly in the left inguinal region. Liver dulness from the sixth rib to the costal margin in the right mammary line. No enlargement of the spleen could be made out, and the examination of the blood for the plasmodium malariae was negative. Urine: sp. gr. 1021; slightly albuminous; no tube casts. The stools were frequent, watery, dark brown, and contained many particles of mucus and a little blood. Microscopical examination showed pus and red blood corpuscles, and large swollen fatty epithelial cells. No amœbæ were found at the first examination. The patient was ordered rest in bed, a milk diet, and the Hospital diarrhœa mixture.

May 2d. Temp. 100°–102° F.; three watery, brown, liquid stools in twenty-four hours.

May 3d. One stool, soft, light yellow and partially formed, without mucus or blood. The cough was troublesome to-day, and there was a little mucoid expectoration.

May 6th. There was pain of a lancinating character in the right side and shoulder. Upper limit of liver dulness in front at the fifth intercostal space, in mid-axilla at the sixth rib, behind at the tenth rib. Edge of liver not palpable.

May 8th. Temp. 101°–103° F.; there was moderate sweating, with pain over the scapula and lower ribs on the right side. Sputum very viscid, mucoid, and of a brownish-gray color. From the angle of the right scapula to the base of the right lung, impaired and slightly tympanitic resonance, increased tactile fremitus, and enfeebled distantly tubular breathing with increased vocal resonance and a few fine crepitant râles.

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May 11. Since May 3d he had had only one stool daily, partially formed and of a yellowish-brown color. There was fever of an irregularly remittent type, ranging from 99° F. in the morning to 102° F. in the evening. Abdomen still soft, and tender to pressure along the ascending and transverse colon. Tenderness also in the sixth and seventh right intercostal spaces in the anterior axillary line, with pain on deep inspiration. High-pitched resonance on the right side from the third intercostal space to the upper border of the fifth rib, extending into lower axilla. Below the fifth rib, dulness to the costal margin. Posteriorly, from the middle of the right scapula to 3 cm. below its angle a sub-tympanitic resonance, and below this point complete dulness. On auscultation, below the right nipple a to-and-fro friction rub; behind, diminished respiratory murmur as far as the middle of the right scapula; at this point a limited area over which there was tubular breathing, with somewhat nasal voice sounds and fine crepitant râles. At the extreme right base, tactile fremitus absent and breathing scarcely audible. Left lung normal.

May 13th. At 9 p. m. he had a violent fit of coughing, followed by vomiting and the expectoration of a quantity of greyish gelatinous frothy material, mixed at first with a considerable quantity of blood, and having a peculiar odor, not sour or gangrenous. Microscopic examination of this material showed pus and blood corpuscles, swollen and fatty alveolar epithelium, and buccal epithelium; no tubercle bacilli. He sweated freely and complained of very slight pain in the region of the liver, but rested quietly after a subcutaneous injection of morphia. On the following morning he was still coughing up in smaller quantities a similar material slightly tinged with blood, but he stated that he felt much better as if something tight inside his chest had given way.

Examination showed the following condition:—A clear percussion note in front over the right lung as far as the fifth rib; from the fifth to the seventh impaired resonance, extending into the lower axilla; below this point complete dulness to the costal margin. A friction-rub was felt below the nipple. Posteriorly the resonance was as noted on May 11th, but tactile fremitus had returned at the base of the right lung. On auscultation, in front, a high-pitched respiratory murmur, with a friction-rub heard below the nipple and in the lower axillary region, and loud gurgling râles in the seventh and eighth

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intercostal spaces. Behind, numerous mucous râles over the lower three-fourths of both lungs; feeble respiration at the extreme base of the right lung, and tubular breathing between the angle of the right scapula and the spinal column.

May 20th. In the evening he coughed up a larger amount than usual of thick ropy muco-pus mixed with blood.

June 7th. He had lost flesh, and the diarrhœa, which had subsided entirely two days after admission, had recurred to a slight degree from May 21st–23d, and more severely since June 5th. There was still irregular fever, and cough which was usually paroxysmal and was followed by the expectoration of thick greyish-red or brown muco-pus. This was found to contain, besides the elements before noted, elastic tissue fibres, fatty degenerated liver cells and bile pigment. Liver dulness, in the median line from the root of the ensiform cartilage vertically downwards 5 cm., in the right mammary line from the sixth rib to 1½ cm. below the costal margin, in the mid-axillary line from the seventh rib to the costal margin, and behind the upper limit was at the tenth rib. No enlargement of the liver could be detected. An exploratory aspiration in the lower right axillary region was negative.

June 12th. Amœbæ were found in the expectoration, some in active motion, others quiescent.

June 14th. Amœbæ were again demonstrated in the expectoration in large numbers and moving very actively. They were most abundant in the more purulent portions of the sputum, and were readily found by placing a small piece of this material on a slide, and gently pressing a cover-glass over it, so as to secure a thin and uniform layer of the material. With a Zeiss D or Leitz No. 7 their movements could readily be followed, and minuter details of structure were studied with a homogeneous oil-immersion lens. The stools varied from four to twelve daily, were of the consistency of thick gruel, light brown in color, and contained particles of muco-pus, in which moving amœbæ were found. From three to four amœbæ were observed in each field examined. They were smaller in size than those seen in the sputum, but in other respects similar to them.

June 16th. Transferred to the surgical ward for exploratory operation. Under an anaesthetic, an incision 8 cm. long was made over

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and in the direction of the eighth rib in the right axillary line, and the cartilages and a portion of the eighth and ninth ribs were removed. The liver was then exposed, the parietal peritoneum sutured to the superficial tissues all around the edges of the wound, and iodoform gauze was tamponed under the edges of the wound to excite adhesions between the liver and abdominal wall.

June 18th. Under morphia the liver, which felt abnormally soft, was aspirated in various directions through the operation wound, with a negative result.

June 20th. There was still cough and expectoration, the diarrhœa was severe, and there was irregular fever ranging from 100°–102° F.

June 21st. Sound passed up through the wound around the upper and lateral surfaces of the liver; no adhesions were detected between the liver and the diaphragm.

June 25th. A second aspiration was made without reaching pus, and the next day an aspiration was made through the back with a similar result.

June 28th. There was a metallic note on percussion between the sixth and seventh ribs in the axilla. Under chloroform a portion of the seventh rib was resected, and on cutting through the parietal pleura more than a litre of thick viscid pus was discharged, in which very many actively moving amœbæ were found, in all respects similar to those found in the sputum. After the operation the patient's pulse improved, and he felt more comfortable. He was, however, extremely weak, and died of exhaustion at 8.30 a. m., June 29th.

Autopsy.—Made at 12 p. m. The organs removed and examined the following morning.*

The anterior border of the right lung free from adhesions. There are firm adhesions between the lung and pleura just outside of the nipple line, and between the lower lobe of lung and the diaphragm. On removal of the lung a straight line from the base of the lung to the apex marks the lateral line of adhesion. Back of this line the pleura is covered with a thick firm layer of fibrinous exudation. Occupying almost the entire lower lobe of the lung is a large abscess cavity with ragged necrotic-looking walls which are partially

* In reporting the autopsy only the portion of the report which relates to the dysentery will be given.

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stained with bile. The irregular wall in places extends one to two cm. into the surrounding lung, forming secondary cavities which open into the large cavity and into each other. The lung around the cavity for a considerable distance is dense, solid and tough. Pus can be pressed from all the small bronchi in this part of the lung. Some portions of the lung here seem to be softer than the rest, and a purulent material can be squeezed from it. The abscess opens into the pleura by a large ragged opening, and a large bronchus passes into the abscess. The floor of the cavity is formed by the diaphragm, and communicates by a large opening in this, 4 cm. in diameter, with the liver.

The liver is large and pale. On the superior surface of the right lobe about the middle, there is a thick fibrinous exudation over an area 2×6 cm. passing obliquely upward and outward. Immediately beneath this the liver is intensely hyperaemic and much denser than normal. Over the entire upper surface of the liver there is a small amount of fibrinous exudation. There is an abscess in the lower surface of the right lobe at a point about corresponding to the hepatic flexure of the colon. The outer wall of the abscess is close beneath the surface of the liver at one point only, enclosed by the capsule and a border of liver tissue 1 mm. thick. The abscess is filled with translucent viscid gelatinous pus, enclosing more solid masses. The walls are tolerably smooth, and there is a small, tough, rather transparent area separating the abscess from the liver tissue. Neither the contents nor the wall of the abscess are stained with bile. There is another abscess in the upper surface of the right lobe just beneath the diaphragm which represents rather an excavation into the liver. This is 5 cm. in diameter on its upper surface, where it communicates through the diaphragm with the lung abscess at the opening into the diaphragm, and 3 cm. deep. Around this abscess the liver tissue is necrotic. Its contents could not be determined for they had escaped on removal of the viscera.

The stomach and small intestines are normal. The entire large intestine is dilated, thickened, and covered with numerous ulcers. It contains a thin brownish material. The ulcers in general have a direction across the bowel. They are irregular in shape with wide overhanging margins, so that when the margins are elevated the ulcer appears twice as large as when looked at from the surface. The smaller ulcers contain translucent viscid pus. When this is removed

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the floors of these and of the larger ulcers are smooth and œdematous-looking. Most of them are limited by the muscular coat. Others pass through this, and are separated from the abdomical cavity only by the thickened peritoneum. The transverse colon is the part most affected: in this the ulcers occupy large areas, with only small masses of mucous membrane separating them. There are also small, more superficial ulcers, some of them being mere erosions. The mucous membrane between the ulcers is thickened and œdematous. The bases of some of these larger ulcers are imperfectly divided by septa, showing that they were formed by the confluence of smaller ones. Microscopic examinations made at the time of the autopsy, 12 p. m., showed numbers of actively moving amœbæ in the liver and lung abscesses and in the colon. On the following morning also they were found in these places. The greatest number of them was in the walls of the liver abscess which was just beneath the diaphragm.

CASE III.—*Dysentery, acute. Abscess of the liver. Amœbæ in the stools. Death. Autopsy.*

F. B., aged 27, a fireman on the SS. Karlsruhe, plying between Baltimore and Bremen, was admitted to the Johns Hopkins Hospital, May 18th, 1890. He had been in the tropics in 1880, but not since, and had enjoyed good health up to the time of his present illness. While at sea, two weeks before his admission to the hospital, he had slight epistaxis followed by vomiting and diarrhœa. His evacuations were at first only watery and accompanied by very little pain, but at the end of a week they were very frequent, as many as forty-eight in twenty-four hours, and contained blood and mucus. With the increased severity of the diarrhœa he had suffered from tenesmus, abdominal pain and occasional vomiting which was sometimes bloody. He had no desire for food, had lost a great deal of flesh, and felt very weak. On the day previous to his admission he had had a slight cough with a little viscid expectoration.

Physical Examination.—Much emaciated; cheeks and eyes sunken; face flushed; tongue clean, of a bright red color, and dry in the centre; skin sallow, dry and harsh; mind clear. Temp. 99.2° F.; pulse 108, dicrotic; resp. 30. Heart sounds a little weak at the apex. At the bases of both lungs there were a few crepitant râles. Abdomen flat and not resistant; distinct sensitiveness to pressure along the course

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of the colon, but no fulness or thickening of the bowel to be felt. Slight rigidity of the upper part of the rectus abdominis on the right side. A few indistinct dark red papules on the abdomen and sides of the thorax, which disappeared on pressure and returned slowly. They were smaller and darker than the papules of typhoid fever, and were not surrounded by an areola.

Liver dulness from the sixth rib to the costal margin in the right mammary line. Spleen dulness from the ninth intercostal space to the eleventh rib; spleen not palpable. Stools partly liquid, partly solid, greenish, very fœtid, and containing much shreddy necrotic-looking material, mucus and here and there blood.

A diet of milk and egg albumen was ordered.

May 19th. Nine stools in the past twenty-four hours, similar in appearance to those observed on the day of admission. Various portions of these were examined and found to contain fragments of elastic tissue fibres, pus and swollen epithelial cells, red blood corpuscles, and a considerable number of actively moving amœbæ, some of which contained one or more red blood corpuscles. The amœbæ were found in all portions of the stools examined, but were most abundant in small greyish-white gelatinous masses floating in the more liquid portions of the stools.

The patient complained of hiccough, which was relieved by sinapisms to the epigastrium.

May 22d. Temp. 100°–100.7° F.; pulse 108. There was no vomiting, but still slight hiccough and much restlessness. Morphia was given subcutaneously at bedtime. Amœbæ were present in the stools, fewer in number and less active.

May 23d. Rectal injections of bichloride of mercury 1–5000 were ordered to be given twice daily, to be retained ten minutes, and the quantity used to be regulated by the patient's ability to retain the injection.

May 25th. The strength of the injections was increased to 1–3000.

May 26th. From May 22d to May 25th there were eight to ten stools daily, and he was failing rapidly. Between five and six o'clock on the evening of June 25th he passed 125 cc. of blood per rectum, and became collapsed. The temperature fell to 97° F., and the pulse became very feeble and thready. A reaction occurred at 3 a. m., and the temperature rose to 103° F. On the following morning the tem-

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perature was 103° F., the pulse 160, there was profuse sweating, the extremities were cold, and there were frequent involuntary evacuations containing numerous sloughs, and, on microscopic examination, large numbers of amœbæ. The patient became completely comatose during the afternoon and died at 11 p. m.

Autopsy.—The entire upper surface of the liver is adherent to the diaphragm. It extends 10 cm. below the ensiform cartilage in the middle line, and is adherent to the anterior abdominal wall. The adhesions are partly fibrinous, partly of newly formed connective tissue; they are easily separated and can be removed as a thin membrane from the surface of the liver. Between and around the adhesions there is a small amount of tough, pale greyish fibrinous exudation. The spleen is also adherent to its surroundings by soft, easily broken down adhesions.

The lower surface of the liver is firmly adherent to the hepatic flexure of the colon and to the stomach and duodenum. On separating these adhesions there is a good deal of tough, greyish fibrinous exudation between them. Below the transverse colon the peritoneum is smooth and glistening, free from adhesions and somewhat congested.

There is an abscess cavity between the lower surface of the liver and the adjacent viscera which are here adherent to the liver. The cavity is bounded by the lower surface of the liver, the stomach, the beginning of the duodenum, and the colon. In the duodenum, 2 cm. from the pylorus, there is a round, smooth opening, 1½ cm. in diameter, communicating with the abscess, and lower down, 2 cm. from this, is another smaller opening. The abscess cavity contains a quantity of greyish translucent pus. Its inner surface is irregular, ragged, and shreds of necrotic tissue project into it. The abscess covers the liver over a space 10 cm. in diameter, and extends 4 cm. into it. The gall bladder forms part of the wall of the abscess, and there is a small opening between it and the abscess. It contains the same greyish translucent pus as the abscess. The liver tissue adjoining the abscess is in several places infiltrated with pus, forming small cavities which communicate with the abscess. There is no sharp line of demarcation separating the abscess from the adjoining liver tissue, and no distinct zone of fibrous tissue. The liver tissue generally is pale and the lobules are not distinct.

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The stomach and small intestine show no change other than the opening between the duodenum and the abscess, which has already been mentioned. The colon is somewhat dilated, the peritoneal surface, apart from the adhesions, is smooth, hyperæmic in places, and feels thickened. The average circumference is 14 cm. It contains a large amount of yellowish-brown, tolerably soft material. Here and there in this material there are small whitish translucent masses, which do not mingle with the other contents and may be lifted out entire. The entire gut is very much thickened. The mucous membrane is pale greyish, œdematous-looking, and there are numerous ulcers of various size, particularly in the ascending and transverse colon. These ulcers have ragged, greatly undermined edges, and generally extend down to, and in some places into, the muscular coat. Between the ulcers there are numerous elevations on the surface of the mucuous membrane, some round, others long and irregular. They project from one to two mm. above the surface. In most of them there is a small opening on the apex, and on cutting into this a cavity filled with viscid greyish gelatinous material is found beneath the mucous membrane. Many of these cavities communicate with one another by means of fistulous tracts beneath the mucous membrane. Some of these tracts may be opened up for a distance of from 3–5 cm.

Actively moving amœbæ were found in the contents of the abscess, in the purulent material, in the gall bladder, in the necrotic tissue, in the wall of the abscess, and in the infiltration of the liver tissue around the abscess. They were abundant in the contents of the colon, particularly so in the gelatinous masses in the bases of the ulcers.

CASE IV.—*Dysentery of a mild type with intermissions. Consolidation at the base of the right lung. Cough with expectoration of bloody muco-pus containing elastic tissue fibres and amœbæ. Amœbæ not found in the stools. Physical signs pointing to deep-seated excavation in the lower lobe of the right lung. Exploratory aspiration of the right lung with a negative result. Operation; abscess involving the liver and the lower lobe of the right lung. Death. Autopsy.*

G. H., aged 28, was admitted to the Johns Hopkins Hospital, June 9th, 1890. He had lived in Baltimore for five years, was a stevedore

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and had been working for some time at the place as the man whose history is given in Case II. Shortly after arriving from Germany, five years before, he had been ill six or seven weeks, and from the account he gave it was probable that his illness was malarial fever. Since then he had been quite well until three weeks ago, when he had a severe fit of coughing lasting all night, followed a few days later by shortness of breath and pain in the right side of his chest, which was increased by coughing or deep inspiration. With the cough, which as well as the pain in the side had abated considerably, he had expectorated some yellowish mucus, but no blood. For a week he had had, every afternoon at four or half-past four o'clock, a chill followed by fever, sweating, and severe headache, and during this time he had also suffered from pain in the right shoulder. He had lost 10 lbs. in weight in three weeks. There was nothing in the account which he gave pointing to any grave intestinal trouble. There had been at times some abdominal pain, and his bowels had been irregular, sometimes moving several times a day, at other times not at all. He had not noticed any blood or slime in his fæces, which were usually solid and well-formed, and could assign no date as the beginning of what was apparently a trivial intestinal disorder.

Physical Examination.—Nutrition good; dorsal decubitus; expression dull and apathetic; face flushed; tongue covered with a brownish-white fur; free sweating. Temp. 101° F., pulse 108, resp. 48. There was some dyspnœa, and when lying on his right side the patient complained of pain in that side. Thorax: both sides of the thorax were symmetrical and expanded equally. Over the right lung in front a clear percussion note to the fifth intercostal space; in the extreme lower and outer mammary, and the lower two-thirds of the axillary region, dulness; posteriorly resonance normal as far as the angle of the scapula, and from this point to the base of the lung, dulness. In the posterior axillary line and to a short distance on either side of it the dulness was absolute. Tactile fremitus absent over the whole area of dulness, and increased immediately above this, as compared with the opposite side. On auscultation, normal respiratory murmur in front as far as the nipple; below this and in the lower two-thirds of the axillary space, feeble breathing, which was distantly tubular in the last-named area: voice sounds just audible. Posteriorly, normal breathing to the middle of the scapula, and from

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this point breathing gradually enfeebled, becoming distantly tubular at the base of the lung. No adventitious sounds were heard, and the voice sounds had not a nasal quality. Left lung, normal.

Heart: apex-beat in the fifth intercostal space, immediately to the left of the mammary line; area of cardiac dulness not appreciably altered from the normal; the second sound reduplicated at the left sternal margin.

Abdomen soft, not distended, sensitive to pressure only immediately below the right costal margin. Over the abdomen, particularly in the groins, there was an indistinct very fine papular rash; the individual papules were very small, and faded, but not entirely, on pressure. Splenic dulness from the eighth to the eleventh rib. Liver dulness apparently extended from the fifth intercostal space to 2.5 cm. below the margin of the ribs in the right mammary line; edge of liver indistinctly palpable.

Cough frequent, hacking, and occasionally paroxysmal; expectoration viscid, reddish-brown or bright red, copious. At the first examination there were found pus and blood corpuscles, and round swollen epithelial cells; no tubercle bacilli.

A light diet and a sedative cough mixture were ordered.

June 11th. No movement of the bowels. Irregular fever with much sweating. Blood examination for plasmodium malariae negative.

June 16th. There was still absence of tactile fremitus with dulness on percussion in the right axilla and at the base of the right lung, and crackling râles were heard during inspiration.

June 24th. Elastic tissue fibres found in the sputum; no tubercle bacilli.

June 17th. Sputum more copious, muco-purulent, reddish-brown. The liver dulness extended 3 cm. below the margin of the ribs in the right mammary line; there was no obliteration of the intercostal spaces, nor any alteration of the shape of the thorax.

June 29th. Actively moving amœbæ found in the sputum by Dr. Reese. There was slight looseness of the bowels from the 25th to the 28th, but no amœbæ were found in the stools.

July 3d. Temp. 99°–101.5° F.; pulse range 90–110; resp. 24–40. Repeated examinations confirmed the presence of amœbæ in the expectoration. An exploratory aspiration was made in the tenth

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intercostal space 9 cm. from the vertebral column; the needle was passed in three directions, but no pus was obtained.

July 6th–12th. Bowels again loose; there were from four to ten watery, brownish-yellow movements daily, containing no blood and very little mucus.

July 13th. Patient transferred to the surgical ward for exploratory operation.

July 14th. An aspiration was made over the anterior surface of the liver with a negative result. The needle was then inserted between the eighth and ninth ribs in the right axillary line, and a small quantity of thick pus was obtained.

On the following day, under anaesthesia, a portion of the eighth and ninth ribs in the posterior axillary line was resected, and on cutting through a little more than 1 cm. of dense connective tissue 250 cc. of thick bloody pus were discharged, in which numerous amœbæ were found.

The abscess cavity extended upward toward the lung and not downward in the direction of the liver. Two drainage tubes were inserted and an absorbent dressing was applied. The patient reacted favorably; during the afternoon the bowels were moved three times and there was slight coughing without expectoration.

July 16th. Abscess cavity irrigated with a solution of quinine 1–5000.

July 17th–31st. Free discharge from operation wound; diarrhoea persistent.

August 8th. Very profuse discharge of pus from abscess. There was much prostration, and weakness.

August 12th. Abscess cavity explored with the finger; this was followed by the escape of much cheesy-looking blood-stained pus, in which inactive amœbæ were found.

August 16th. During the last two days the patient had been breathing with difficulty. He died at 3 p. m.

Autopsy.—The colon shows numerous ulcers which are especially marked in the transverse and descending portions. These ulcers are sharply circumscribed and of various shapes, with the long axis generally transverse to the axis of the intestine. Most of them extend to the muscular coat and the edges are greatly undermined. In the bases of some of them there is some erosion of the muscular coat. One or

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two of the ulcers extend through the transverse muscular coat which is dissected up for a short distance around the ulcer.

There is an abscess of the liver on the upper surface immediately beneath the diaphragm. It measures 6 × 8 cm. The walls are ragged and necrotic, the necrotic tissue extending deeply into the surrounding liver structure.

The diaphragm is not perforated. The lower lobe right lung is intimately adherent to the diaphragm. Almost the entire lobe is occupied by an abscess cavity with ragged necrotic walls. The lower portion of the upper lobe is of a pinkish-grey color, gelatinous-looking and airless.

CASE V.—*Dysentery. Amœbæ in the stools. Recovery.*

Jos. B., aged 41, was admitted to the Johns Hopkins Hospital June 23d, 1890. He had lived in America for two years and had never been south of Baltimore, where he was engaged as a stevedore, working in the same locality as Wm. K. and G. H. (Cases II and IV). Nine weeks before admission to the Hospital he had been suddenly seized with diarrhœa and cramps in the abdomen. The movements were watery and frequent, as many as sixteen in twenty-four hours. He had had no chills, no epistaxis, very little fever, and had not noticed any blood in the evacuations. At the beginning of his illness he had vomited once, and again a few days before admission. After the fourth or fifth day the diarrhœa, though persistent, had been painless as a rule, though occasionally when turning on his side he would have slight abdominal pain. He felt very weak and had taken very little food during the last few days.

Physical examination.—Not emaciated, skin of a sallow earthy color, tongue heavily coated. Temp. 102.2° F., pulse 90, resp. 25–30. Thoracic organs normal. Abdomen not distended, soft; slight tenderness to pressure in the epigastrium, and some gurgling but absolutely no tenderness along the course of the colon. The liver dulness in the median line extended from the middle of the ensiform cartilage 6 cm. vertically downwards, in the right mammary line from the sixth rib not quite to the costal margin, and posteriorly its upper limit was at the eleventh rib. Splenic dulness from the eighth to the eleventh rib: edge of spleen palpable. Blood examination negative for plasmodium malariae. Diet of milk and egg albumen.

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June 24th. Five stools in twenty-four hours, very liquid, brownish-yellow, containing very little mucus or blood, and some small flakes of greyish-yellow muco-pus. In these last mentioned portions of the stools very many actively moving amœbæ were found on microscopic examination.

June 25th. Six stools: amœbæ present. Injection of bichloride of mercury, 1–5000.

June 26th. Injections continued.

June 30th. Left hypochondrium quite sensitive to pressure. No increase in the area of liver dulness.

July 4th. Nitrate of silver was ordered internally. Five stools in twenty-four hours, which contained a few amœbæ.

July 5th–7th. Only one movement daily, each containing some formed fæcal matter.

July 10th. Convalescent: tongue slightly furred, skin moist, no abdominal pain. No movement of the bowels since the 7th.

July 19th. One watery stool: slight tenderness on pressure in the right iliac fossa, and in the right hypochondrium. No enlargement of the liver.

July 22d. Discharged well.

CASE VI.—*Dysentery, acute. Amœbæ very numerous in the stools. Perforation of the bowel. Death. Autopsy.*

Maggie F., aged 47, was admitted to the Johns Hopkins Hospital July 3d, 1890. She had lived in America for twenty-eight years, had been ill with typhoid fever when a child and with pneumonia some years ago. Five weeks before admission she had been working on a farm a few miles south of Baltimore, and at the end of the first week had suffered from profuse diarrhœa and severe abdominal pain. The stools were very frequent, small, accompanied by much straining, and consisted of mucus mixed with blood. Since that time she had had a persistent diarrhœa, with pain in the abdomen, slight fever, intense thirst and loss of appetite and strength. The drinking water used on the farm was drawn from a pump and a spring, and the water from the former was described by her as "heavy and bad."

Physical examination.—Not emaciated, but much exhausted; face flushed, skin moist, tongue furred and moist. Temp. 98° F., pulse 108 bounding, resp. 36.

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Thoracic organs normal. Abdomen a little distended, exquisitely sensitive to pressure in the flanks and iliac regions. Liver dulness within normal limits, and spleen not palpable.

In the first twenty-four hours there were ten loose stools, accompanied by severe abdominal pain and tenesmus. They varied much in appearance, some being pultaceous and of a mottled greenish-white color; others liquid, brownish-red, and containing streaks of bright blood, blood-stained mucus, and tough, ragged, yellowish-grey, necrotic-looking material; others again being small, gelatinous, translucent and mixed with more or less blood. Actively moving amœbæ were found in great numbers. The urine was slightly albuminous.

Diet of egg albumen and barley water. Injections of ice-water, one litre, to be followed by starch and laudanum enema.

July 4th, 5th, 6th and 7th. On these days there were respectively eleven, nine, seven and eight stools, of a similar appearance to those noted above, all containing amœbæ. The temperature had been rising gradually since July 3d, and now ranged from 100° to 101° F.

July 8th. At 9 a. m. she complained of a sudden intense pain in the abdomen, which on examination was found to be a little more distended and generally very sensitive to pressure. There was no vomiting, but the pulse became small and more frequent. She appeared to be suffering intensely and was bathed in perspiration. Perforation of the bowel was diagnosed, and morphia was given subcutaneously in quantities sufficient to allay the pain. In the stools examined during the day there were found, in addition to numerous amœbæ of a smaller size than those before observed, necrotic masses containing elastic tissue fibres. One of these fragments measured 10 cm. in its longest diameter.

July 9th. Death occurred at 1.30 a. m.

Autopsy.—On opening the abdominal cavity there was a free escape of gas. The entire surface of the peritoneum is cloudy and covered with a slight fibrino-purulent exudation. It is hyperæmic and there are a few small ecchymoses scattered over it. The small intestines are lightly adherent to one another, and between the adhesions there is a small amount of fibrinous pus. A number of folds of small intestine are more firmly adherent to one another and to the transverse colon at the hepatic flexure. In the upper part of this adherent mass

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there is a small opening leading directly into the colon. On breaking up the adhesions a large opening is found in the colon which is partly covered by a dark slough and is almost entirely closed by the adhesions. In the peritoneal cavity there are 1000 cc. of dark fluid with a strongly feculent odor.

Stomach and small intestines show no change. The large intestine contains brownish fluid contents. Its walls are very much thickened. The fat in the meso-colon is hyperæmic, and the lymph glands are enlarged and soft. In the ascending colon there are several small bends which are adherent to each other. There is very extensive ulceration of the mucous membrane, the ulcers varying greatly in character and extent. In the lower portion of the descending colon there are some small ulcers which are filled with greyish viscid gelatinous pus. On removing this the base of the ulcer appears clean and extends down to the muscular coat. The edges are greatly undermined. In the upper portion of the descending colon there are larger ulcers with deeply undermined edges and a clean base extending down to the muscular coat. Most of these have their longest diameter transverse to the axis of the bowel. The undermined edges are filled with gelatinous pus, and some of the ulcers are connected with one another by means of sinuous tracts as in Case III. In the beginning of the transverse colon the entire gut is involved in a large loss of substance 8 cm. in length. This ulcer is partly covered by large masses of black or brownish necrotic material. Most of this is one large mass attached to one side of the ulcer. The under surface of the slough has a distinctly striated appearance. Partly covering this slough, and here and there attached to it and extending beneath the undermined edges, there is a large amount of greyish gelatinous-looking pus. The ulcer varies in depth. In some places it is formed by the transverse muscular coat, and in others again it extends below this. The large adherent slough is apparently composed of the transverse muscular coat, and on elevating this, one of the longitudinal bands of muscle is visible extending along the entire base of the ulcer. A large portion of the base is formed by the thickened peritoneum. The undermining of the edges takes place beneath the muscular coat just as beneath the mucosa, and the different depths of ulceration are preceded by a dissection of the coat immediately beneath. The ascending colon forms an irregular

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mass due to the adhesions around it and between the folds. Just at the hepatic flexure and covered in by the adhesions, there is a large opening 10 × 3 cm. The wall of the gut here is formed partly by the adherent ileum and partly by a large necrotic slough of the muscular coat, which adheres along the lower end of the ulcer. The entire gut, for a distance of 17 cm., is involved in the ulcer. This ulcer has the same general appearance as that in the transverse colon, but is in every way more extensive. The greater part of the ulcer is covered with the large black or brownish slough spoken of. The upper surface of this slough is ragged and fluffy. The lower is smooth and striated transversely, and is plainly the entire transverse muscular coat. In the cæcum and beginning of the ascending colon there are smaller though extensive ulcerations of the same character as the others. In the rectum there is also an extensive ulcer, which begins 2 cm. above the anus and extends 9 cm. upward, involving the entire calibre of the gut. It is covered with adherent sloughs; its edges are undermined, and it has all the characters of the other ulcers. The mucous membrane everywhere adjoining the ulcers, is pale, swollen and œdematous-looking. The pus in the ulcers is very gelatinous and viscid. It can be removed in large drops and remains in a solid mass beneath the cover-slip, having no tendency to spread out.

Microscopic examination of the peritoneal adhesions, and of the fluid contained in the cavity, shows numerous bacteria, both micrococci and bacilli, pus cells and large swollen cells probably derived from the endothelium. No amœbæ were found in this. Pus taken from the ulcers shows numerous actively motile amœbæ.

CASE VII.—*Dysentery of two weeks' duration. Amœbæ in the stools, disappearing during convalescence. Recovery.*

Gustav S., aged 27, stevedore, was admitted to the Johns Hopkins Hospital July 4th, 1890. He had lived in Baltimore for three and a half years, working most of this time on the wharves in the same locality as Wm. K., G. H., and Jos. B. (Cases II, IV and V), but for ten days preceding his illness he had lived at Sparrow's Point, Maryland, and had obtained his drinking-water from a well which, according to his account, was foul. During the previous summer he had been ill for a week with fever and vomiting, but without looseness of the bowels, and from that time until two weeks before

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entering the Hospital his health had been good. At this time he was attacked with diarrhœa and nausea without vomiting. The stools had been painless at first and frequent, especially at night, as many as fifteen in twenty-four hours, and had contained a good deal of blood and greenish material. Notwithstanding the diarrhœa he had kept on working until June 30th, when he took to his bed feeling quite weak. For a few days he had had severe abdominal pain before going to stool with straining during the act, and at the time of his admission he could not lie on his left side without suffering pain. He was continually craving fluids, and suffered from persistent nausea, though unable to vomit, and taking very little food.

Physical examination.—Nutrition and general condition good, skin of an earthy color, tongue indented and heavily coated. Temp. 99.2° F., pulse 60–70, resp. 20. Thorax negative. Abdomen not distended, soft, slightly tender on pressure in the lower zone, tympanitic throughout. Splenic dulness from the eighth intercostal space to the eleventh rib. The liver dulness extended from the upper border of the sixth rib to the seventh rib in the right mammary line, and below this the percussion note was tympanitic; posteriorly and in the axilla the outline of hepatic dulness was normal. There were three movements of the bowels in the first twenty-four hours, all very liquid, almost homogeneous, yellowish-brown, containing a little dark blood, mucus, small fragments of shreddy material, and a moderate number of amœbæ.

Diet of milk and egg albumen. Injections of ice-water twice daily.

July 7th. Seven stools like those described above, containing very many active amœbæ.

July 12th. Injections of sulphate of quinine, 1-5000, substituted for the ice-water injections: two such injections to be given daily.

July 14th. Three stools containing amœbæ. During the first week the temperature had ranged between 98° and 101° F.; it was now from 98°–99° F.

July 19th. Nitrate of silver was given internally.

July 23d. During the four preceding days there was only one movement of the bowels daily, which contained some solid fæcal matter and in which no amœbæ were found.

August 5th. He had gained three pounds in weight since his admission, and was feeling quite well. There were from one to two

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stools daily, soft, partially formed, homogeneous, without blood, and with only a trace of mucus. He was discharged on this day.

CASE VIII.—*Dysentery, acute, preceded by indefinite gastric symptoms. Stools consisting of blood, mucus and shreddy detritus; no amœbæ found. Intestinal hæmorrhage. Death. Autopsy. Amœbæ found in sections of the intestinal ulcers.*

J. D., aged 27, was admitted to the Johns Hopkins Hospital, August 18th, 1890. As he spoke no language but Russian, it was impossible to obtain an accurate account of his illness from himself, but it was ascertained that he had worked at Sparrow's Point, Maryland, and that twelve weeks before entering the hospital he had had uneasiness, a sensation of distention and some pain in the epigastrium. Nothing further had occurred until a week before admission, when he began to suffer from diarrhœa, accompanied by severe cramps in the abdomen and straining at stool. The movements had been frequent, from twenty to twenty-five daily, and had contained blood and slimy matter. He had not vomited. His illness was attributed by his friends to the use of bad meat.

Physical Examination.—General condition good; weight 142 lbs. Cheeks flushed, skin dry, tongue almost clean, moist. Temp. 99.2° F.; pulse 96, of medium volume; resp. 24. Thoracic viscera normal. Abdomen retracted, not sensitive to pressure. Spleen not palpable. The liver dulness did not quite reach the costal margin. Urine albuminous, containing a few hyaline tube-casts. There were six stools in the first twenty-four hours, containing large and hard curds of milk, much bloody mucus, bright blood, and greenish pultaceous material. On microscopic examination there were found blood and pus cells, large oval or round epithelial cells containing fat drops, and much amorphous material: bacteria were not numerous, and no amœbæ were found.

Diet of egg albumen exclusively.

August 25th. Since admission the temperature has been generally sub-normal, with the exception of a daily rise to 100° or 101° F. at 4 p. m.; pulse 132, small. Expression dull, emaciation marked; abdomen much retracted but nowhere sensitive. Stools 5–10 daily, occasionally involuntary, accompanied by pain in the abdomen and tenesmus: some of the movements were similar to those first observed,

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while others were very liquid and brownish-red, containing small dark red blood clots and looking like meat-washings; all of them were horribly offensive. During the first three days ipecac, guarded by a small dose of opium, was given according to the method used in India, without materially affecting the number or the character of the evacuations. Subsequently injections of starch and laudanum were employed.

August 27th. At 8 p. m., hæmorrhage from the bowels, 125 cc. of coagulated blood. Two hours later the patient was collapsed: pulse 132, filiform; resp. 36; extremities cold and clammy. He died during the evening.

Autopsy.—The stomach and upper part of the small intestines are normal. In the upper third of the ileum there are patches of congestion which become continuous in the lower half of the gut. Patches of very superficial necrosis of the mucous membrane are found, commencing 100 cm. above the valve and extending nearly to it. They correspond to the areas of most intense congestion.

There is more or less extensive ulceration throughout the large intestine. It is least marked in the transverse colon, but everywhere else the bowel is riddled with ulcers. The mucous membrane, where intact, has a grayish-red color deepening to a chocolate brown. There is nowhere any necrosis or fibrinous exudation on the surface. The ulcers themselves are very irregular in shape; they run into one another, and are either clean or covered with a very thin granular-looking pellicle of a grayish color. They often reach the muscular coat but do not penetrate it. Their edges are greatly undermined. In the transverse colon there are only a few widely separated ulcers, and between these the mucous membrane is healthy. In the cæcum the ulcers are shallower than those in the other parts of the intestine. The submucous coat in some places is considerably thickened.

CASE IX.—*Dysentery beginning as a mild transient diarrhœa, followed by alternate constipation and looseness of the bowels. Severe pain in the right side with vomiting; after an interval of some days, cough, with expectoration of bloody tenacious muco-pus. Enlargement of the liver; physical signs pointing to deep-seated cavity in the lower lobe of the right lung. Amœbæ and elastic tissue fibres*

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found in the sputum; amœbæ subsequently found in the stools. Diminution of cough and expectoration, and disappearance of amœbæ from the sputum and stools. Return of cough with profuse expectoration containing a few amœbæ. No return of diarrhœa; amœbæ not found in the stools. Subsidence of cough and disappearance of amœbæ from the sputum. Patient discharged much improved.

William W., negro, aged 29, admitted to the Johns Hopkins Hospital October 5th, 1890, was born and had spent most of his life in Baltimore. He had suffered from malaria several years before, from inflammatory rheumatism nine years ago, and three years later from chancre and inguinal buboes. He had lived in the West Indies for fifteen months about six years ago, and during this time had not lost a day's work from ill health. Toward the end of April, 1890, he had a slight attack of diarrhœa lasting four days, which he attributed to drinking beer, and on May 1st he left Baltimore to go to Flowertown, near Philadelphia. During the first two weeks of his stay there his bowels were very constipated, and sometime after he had severe pain shooting round from the spine to the right side with nausea and vomiting. He entered a hospital in Philadelphia about the end of June, and there, still suffering from pain in the right side, began to cough and to spit up much thick bloody phlegm. During the three months he spent in hospital this condition persisted, and his bowels were very irregular. He would have no movement for two or three days, then two or three stools, painless, soft, yellowish or greenish, occasionally containing a little blood. He also had much pain in his right shoulder which prevented him from lying on that side. About September 1st he left Philadelphia, still suffering from irregular diarrhœa, cough, fever and occasionally sweating. He had lost 29 lbs. in weight since May 1st, but retained a good appetite and slept well when not disturbed by coughing.

Physical Examination.—Powerfully built and still well-nourished; weight 175 lbs.; dorsal decubitus; occasional paroxysms of coughing. Temp. 100.4° F.; pulse 84, dicrotic; resp. 28. Chest well-formed; measurements and expansion equal on the two sides in the upper half of the chest—in the lower half the left side expands slightly more than the right; breathing largely diaphragmatic. Normal resonance over

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the right lung in front to the sixth rib, in the axilla to the fifth rib; from the fifth to the seventh rib high-pitched resonance. Posteriorly, normal resonance to the lower border of the spine of the scapula; below this point to the angle of the scapula defective resonance, and at the base absolute dulness with absence of tactile fremitus. Left lung resonant throughout. Auscultation: over the right lung in front feeble breath sounds in the second and third intercostal spaces, distantly tubular breathing in the fourth intercostal space and in the lower axilla with mucous râles on coughing: posteriorly, enfeebled respiratory sounds over the right lung; from the spine of the scapula to the base of the lung mucous râles at the end of inspiration, becoming more numerous and coarser toward the base; over the area of absolute dulness, as far as the posterior axillary fold, tubular breathing, loud coarse râles and articulate voice sounds. There was some sensitiveness on deep pressure in this region. Left lung normal. Heart sounds clear at apex and base. Abdomen not distended; tender on pressure just below the right costal margin, but not elsewhere. Upper limit of liver dulness at the fifth cartilage in the median line, at the sixth in the right parasternal line, at the lower margin of the sixth rib in the mammary line and at seventh rib in the mid-axillary line. There was sensitiveness to pressure over the lower ribs and the edge of the liver was palpable 3 cm. below the costal margin. The spleen was not palpable. Urine, sp. gr. 1013, very slightly albuminous. Sputa copious, about 400 cc., reddish-brown, mixed with blood, in distinctly isolated muco-purulent masses, surrounded by a clear frothy liquid.

On microscopic examination there were found pus and red blood corpuscles, swollen, fatty and pigmented alveolar epithelial cells, elastic tissue fibres and active amœbæ. Fæces formed, and adhering to them small particles of mucus and a little blood.

October 6th. In mucus adhering to a well-formed stool there were found numerous actively moving amœbæ.

October 12th. Temp. range during the past week 97°–100° F. One or two stools daily, formed. Amœbæ present.

October 18th. Temp. normal. Sputa from 200–300 cc., still slightly brownish red, but more mucoid and without amœbæ. The liver dulness did not now extend below the costal margin: its upper limit was at the sixth rib in the mammary line and at the eighth rib

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at the posterior axillary fold. The respiratory sounds at the base of the right lung were feeble and nowhere markedly tubular or accompanied by loud râles.

October 27th. Marked improvement. Sputa 40–50 cc. One to three stools in twenty-four hours. No amœbæ found in stools or sputum. Weight 167 lbs. Discharged at his own request.

December 4th. Re-entered hospital stating that three days after his discharge he had again had severe cough with profuse blood-stained expectoration, but no diarrhœa.

Physical Examination.—General condition, as on previous admission, excellent. Temp. 98°–101° F., pulse 72, resp. 30. Right lung clear in front and in the axillary space; posteriorly, defective resonance from the middle of the scapula to the base of the lung, where the percussion note was hard and slightly tympanitic. Tactile fremitus diminished at the base of the right lung. On auscultation—respiration normal in front and in the axillary space: posteriorly, feeble breathing in the interscapular region with mucous râles on coughing: from the angle of the scapula to the base absence of respiratory sounds during quiet breathing; distant tubular breathing with râles—not resonant or suggestive of excavation—on deep inspiration; while on coughing the character of the breath sounds was entirely changed, inspiration being distinctly tubular and expiration accompanied by loud resonant râles. Left lung normal. No enlargement of the liver anteriorly. Abdomen negative. Sputa of a sweetish odor, more purulent and less bloody than those first observed: elastic tissue fibres and a few amœbæ found on microscopic examination. Fæces formed; no amœbæ found in them.

December 6th–12th. Temp. 98.5°–100.5° F. Severe cough, usually paroxysmal, with copious expectoration. One to two movements of the bowels in twenty-four hours.

December 14th. From this day until his discharge from the hospital the patient improved gradually. The temperature was normal with the exception of temporary elevations on December 27th and 30th, January 3d, February 9th and April 26th. The expectoration was very variable in amount ranging from 50–840 cc. daily, but it was observed that the purulent and bloody portions progressively diminished in amount, and that on those days on which the expectoration was most abundant it consisted mainly of a thin clear

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slightly frothy liquid resembling saliva. The bowels were irregular, occasionally requiring a laxative, and except under these circumstances the fæces were formed and of normal appearance.

February 27th. Note on the condition of the right lung: defective resonance from mid-scapula to the base. On quiet breathing respiratory sounds feeble and distant over this area; on coughing, deep tubular breathing with a few râles just below the angle of the scapula.

May 10th. Cough very much diminished; general condition very good, weight 181 lbs. Sputa 60–140 cc., frothy, mucoid, occasionally blood stained; no amœbæ found for several weeks. Fæces well-formed containing no blood, mucus or amœbæ.

Discharged to-day much improved.

CASE X.—*Dysentery of five months' duration, commencing with loss of strength, lassitude and slightly bloody stools, and followed by diarrhœa marked by exacerbations. Severe pain in the abdomen and right side of the chest, accompanied by paroxysmal cough, bloody expectoration and profuse sweating. Physical signs of abscess of the liver, involving the base of the right lung. Amœbæ in the fæces and sputum. Death. Autopsy.*

George S., aged 35, entered the Johns Hopkins Hospital, January 12th, 1891. He had lived in Baltimore many years, and with the exception of several attacks of chills and fever, the last of which occurred ten years ago, had been in perfect health until about midsummer, 1890. At this time he had pains in his back, general lassitude, loss of appetite and strength, and occasionally passed a little blood with his stools. He suffered no further inconvenience until the first week of September, when diarrhœa came on, which was severe during two weeks. He suffered no pain, but passed blood and mucus by the bowel, though not in large quantities. Throughout this and the following month the diarrhœa persisted with varying intensity, and about the end of November he began to have severe pain in the abdomen and right side of the chest and about the right shoulder-blade. Coincidentally with this he began to cough and to spit up a blood-stained viscid material. Both the cough and the expectoration were paroxysmal, and his pain was increased when he lay on either side instead of on his back. During the three weeks previous to his admission to the Hospital the diarrhœa had increased in severity, and

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he had as many as fifteen movements in twenty-four hours, which he described as being dark-colored, watery, but containing no blood. He had had no rigors and but little fever, but occasionally had sweated profusely, and had lost much flesh since the beginning of his illness. He stated that he usually drank the water supplied by the city mains, but on several occasions during the summer he had drunk water from a spring which was at times contaminated after rains with surface water coming from the "Spring Gardens," in South Baltimore.

Physical Examination.—Very weak, emaciated, pale and sallow; dorsal decubitus, no dyspnœa, but frequent hacking cough; temp. 98.6° F., pulse 120, resp. 26. Skin dry and rough, tongue brown and dry in the centre. Thorax: slight bulging of the chest-wall behind from the seventh to the ninth rib on the right side, with deficient expansion and partial obliteration of the intercostal spaces. Normal resonance over the right lung in front and in the axillary space; posteriorly, defective resonance from the spine to the angle of the scapula; below this, absolute dulness with tenderness on percussion and pressure, especially at the posterior axillary fold. Tactile fremitus increased at the base of the right lung. On auscultation, normal vesicular breathing in front and in the upper two-thirds of the axillary space; a few small mucous râles in the lower third. Posteriorly, feeble breathing from the apex to the angle of the scapula, with small râles during inspiration: in the infrascapular region, during quiet breathing, breath sounds feeble and distantly tubular with larger and louder râles; they were most marked between the angle of the scapula and the posterior axillary fold. On coughing the tubular quality of the respiration was accentuated. Left lung normal throughout. Heart sounds clear. Abdomen soft and relaxed, slightly fuller in the right than in the left flank; sensitive to pressure, particularly in the epigastrium, about the navel and along the course of the colon. The edge of the liver could not be felt distinctly: the lower limit of hepatic dulness extended in the median line to 25 cm. from the navel, and to the same level in the right mammary line; its upper limit was at the ensiform cartilage in the median line, at the upper border of the sixth rib in the right mammary line and at the lower border of the sixth rib in the mid-axillary line. The spleen was not palpable. Urine scanty and albuminous. Expectoration abundant, very viscid, frothy, of a brownish-red color,

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containing small yellowish-white, soft, cheesy-looking masses. On microscopic examination, pus cells, red blood corpuscles, alveolar epithelium and hæmatoidin crystals. In the sputa examined a few days before the patient's admission to the Hospital, amœbæ and elastic tissue fibres had been found: these were again demonstrated.

Diet of milk and egg albumen. Injections of starch and laudanum.

January 13th. Blood count: R. corp., 3,956,000; W. corp., 53,000 per cub. mm.

January 15th. Temp. 98°–101° F.; thirty-five stools in twenty-four hours, liquid, small, very offensive, containing reddish-brown mucus, soft greenish material and numerous small whitish-grey masses. Active amœbæ were abundant.

January 17th. Nine stools in twenty-four hours. Sputa 170 cc. Quinine injections, 1–5000, bis. die.

January 24th. He had been growing weaker every day, and during the last few days had suffered much pain in the abdomen and the right side. The cough was severe, the expectoration ranged from 75–120 cc., while the stools were reduced to from 3–6 daily and were passed involuntarily. When the patient was not under the influence of morphia the movements were preceded by great abdominal pain. Amœbæ were constantly present in the fæces and sputa. From the 18th to the 24th the temperature ranged from 99°–102° F., the pulse from 120–150, and the respirations from 28–44.

Death occurred on the afternoon of January 24th.

Autopsy.—The right lung is firmly adherent to the diaphragm and slightly adherent along its posterior border. The pleura over the posterior surface of the lung and the corresponding parietal pleura is covered with a thin fibrino-purulent exudation which can be easily removed. The lower lobe of the lung is consolidated in almost its entire extent. The consolidated lung is greyish in color, more translucent and of about the same consistency as in a well marked case of interstitial pneumonia. The cut surface is smooth and pus can be squeezed from the bronchi. In the lower posterior part of the lobe there is a large abscess cavity with ragged necrotic walls which communicates with the pleura and with an abscess of the liver by an opening in the diaphragm. On section of the wall of the abscess there is a line of opaque caseous-looking tissue of variable depth

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which has no sharp limitation toward the surrounding firm solid lung. A large bronchus passes directly into this cavity.

The peritoneum presents a curious appearance. There is no accumulation of fluid. The liver is very large, its lower border being 13 cm. below the ensiform cartilage in the median line and 8 cm. below the costal margin in the right mammary line. There are no adhesions between the loops of intestine in the lower portion of the abdomen. In the upper part the intestines are slightly adherent to each other and to the liver and gall bladder. The entire surface of the peritoneum is cloudy; on passing the fingers over it, it is slightly roughened. Over the entire anterior surface of the liver and over a part of the stomach and transverse colon there is a fibrinous exudation. This exudation does not cling closely to the surface as does the ordinary exudation but can easily be scraped off or even pushed off with the finger. It is opaque and gelatinous. There seems to be considerable fluid enclosed in it. It is most extensive over the liver, the fissure being filled up with it. There is a small amount of it in the pelvis. There seems to be no exudation over the small intestines, at least none can be scraped from them. They appear to be bathed in a more liquid exudation, which has not the character of pus. Between the liver and the diaphragm there is a considerable accumulation of the fibrinous exudation which has been spoken of.

The liver is very large, weight 2600 gms. Over the surface of the right lobe there are numerous round elevated regions of a pale yellowish color which vary in size from 0.5 to 5 cm. in diameter. The smallest of these are not at all or very slightly elevated above the surface. The larger ones are considerably elevated. There are a few such places in the left lobe. In the right lobe just beneath the diaphragm there is an abscess 6 cm. in diameter which communicates through the diaphragm with the abscess of the lung. This abscess has firm fibrous walls, its interior surface is ragged and necrotic, and it contains an opaque gelatinous-like pus. Close to this abscess and about in the centre of the lobe is another abscess of equal size with walls not so fibrous as the first. The liver tissue about both these abscesses is dense and very hyperæmic. Scattered through the liver there are numerous small abscesses corresponding to the elevations noted on the surface. Most of these are immediately beneath the capsule or have only a thin covering of liver tissue separating them

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from this. They vary in size from 2 mm. to 4 cm. in diameter. The larger ones have very irregular walls and have evidently resulted from the coalescence of a number of smaller abscesses. On section the larger of these abscesses are filled with a rather thick gelatinous-looking material in which there are frequently more solid portions. Only a portion of the fluid escapes on section leaving a spongy-looking whitish mass of tissue behind. The very smallest areas represent rather a mass of necrotic tissue than a true abscess. None of them had sharp walls, and, with the exception of the two large abscesses mentioned, beneath the diaphragm and in the middle of the right lobe, there was not a trace of fibrous tissue anywhere about them.

The colon is free from adhesions except at two places where it is firmly adherent to the stomach. At these points, on breaking up these adhesions, there is an entire destruction of the gut which is walled in by the adhesions. The intestine is filled with intensely fœtid brownish contents. There are numerous ulcerations which are most extensive in the cæcum and ascending colon. Here there are large areas entirely devoid of mucous membrane with a firm base extending to the muscular coat. The mucous membrane adjoining these is hyperæmic and undermined. In the transverse colon there are much deeper ulcers—one penetrating down into the fat of the meso-colon, and one passing entirely through to the adherent peritoneum. The entire colon is greatly thickened. The ileum, for a distance of 12 cm. above the ileo-cæcal valve, contains numerous ulcers with undermined edges and clean bases. They present very much the same characters as those in the colon, but are not so extensive. The entire ileo-cæcal valve is ulcerated.

Microscopic examination of the exudation in the peritoneal cavity, and of the pus in the abscesses in the liver and lung shows numerous actively moving amœbæ. They are most numerous in the pus from the smallest abscesses of the liver.

CASE XI.—*Dysentery, beginning with an acute attack and passing into chronic dysentery. Amœbæ in the stools. Patient still under observation.*

Robert D., negro, aged 48, entered the Johns Hopkins Hospital, January 19th, 1891. He had lived in Mexico during the civil war, and since that time in Maryland. He had contracted syphilis twenty

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years before, and in 1873 had a mild attack of small-pox. Six weeks before admission, having previously been in perfect health, he had severe griping pain, localised at first about the navel and subsequently in the lower part of the abdomen, followed by diarrhœa. There were from seven to eight stools daily, small, liquid, offensive, containing blood and accompanied by pain about the anus. He had not suffered from vomiting, nausea or rigors, but occasionally had slight fever and chilly sensations, and though his appetite was unimpaired he had lost about 30 lbs. in weight and felt weak. He passed urine frequently and in small quantity at a time. Nothing suggestive of the source of infection was elicited from him.

Physical Examination.—Well-built and muscular, somewhat emaciated; tongue clean, skin cool and moist. Temp. 98.4° F., pulse 80–90, resp. 24. Lungs normal. Heart sounds clear. Abdomen soft and relaxed; slight sensitiveness to pressure in the epigastrium, none whatever in the course of the ascending and descending colon: in the left iliac fossa the bowel felt thickened and could be rolled under the finger. Upper limit of liver dulness at the upper margin of the eighth rib in the mammary line, at the ninth rib in the mid-axillary line and at the eleventh rib behind. The liver was not palpable in the right hypochondrium, and there was no enlargement of the spleen. The urine contained a trace of albumen. A stool examined on the day of admission was liquid, brownish-red, very offensive, and contained much translucent bloody mucus and a few solid whitish particles. On microscopic examination there were many swollen and fatty epithelial cells, red blood corpuscles and inactive amœbæ.

A liquid diet was ordered.

January 22d. Fluid fæces withdrawn by means of a tube were of a light yellowish-brown color with very little mucus and blood, and no sloughs. Active amœbæ were abundant in the mucus. There were from three to seven such movements daily.

January 28th. Injections of quinine, 1–2500, to be given three times daily.

February 1st. Quin. sulph. 0.3 gm. t. i. d.

February 11th. No movement of the bowels except with the injections; amœbæ not found in the last three stools examined. The injections were discontinued.

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March 12th. During the preceding month there had been from one to three movements daily, of the color and consistency of thick pea-soup (occasionally less fluid), containing very little clear mucus and no blood. A moderate number of active amœbæ were found in the stools on this day, and the quinine injections were resumed.

March 15th. In fæces obtained by rectal tube and examined immediately, a few amœbæ were found. They were pale, circular in outline and motionless.

April 10th. Quinine injections discontinued.

April 24th. Quinine injections, 1–2500 t. i. d.

April 30th. Quinine injections discontinued.

May 4th. Liquid brownish-yellow fæces, containing much mucus and blood-stained material; many very actively moving amœbæ in all portions of the stool.

May 19th. Liquid fæces, *alkaline*, with numerous particles of mucus and blood-stained muco-pus; many active amœbæ.

May 25th. Semi-liquid stool, *acid*, brownish-yellow, intensely fœtid, containing partially formed fæcal masses and undigested food; no amœbæ.

May 26th. In *alkaline* fæces examined yesterday there were a few active amœbæ; in *acid* fæces a few motionless amœbæ.

May 28th. One neutral and one acid stool; many motionless, and a few active amœbæ in both.

May 29th. Acid stool, very few shrunken, motionless amœbæ with coarsely granular protoplasm.

June 6th. Alkaline stool, numerous active amœbæ, rather smaller than usual. Quinine injections, 1–2500.

June 10th. Alkaline stool, a moderate number of active amœbæ.

June 12th. Acid stool, active amœbæ both single and in groups.

June 29th. Slight anæmia and weakness; weight 160 lbs; occasional sweating at night, but no rigors. Temperature usually normal; at irregular intervals, fever not exceeding 100° F. Pulse 70; resp. 18. Heart and lungs normal. Abdomen soft; slight sensitiveness to pressure and gurgling in the course of the transverse colon. No enlargement of the liver. Stool examined June 28th was yellowish-brown, pultaceous, contained much blood-stained muco-pus, and reacted with both red and blue litmus paper. Amœbæ were abundant, as many as forty to sixty in a group, of large size and actively moving.

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Quinine injections, 1–1000. Patient still under observation.

CASE XII.—*Dysentery, of four weeks' duration, preceded by a painless and intermittent diarrhœa lasting nine months. Amœbæ in the stools, disappearing during convalescence. Recovery.*

Gottfried W., aged 11, admitted to the Johns Hopkins Hospital February 17th, 1891, had been ailing for nine months from an intermittent painless diarrhœa. Four weeks before his admission to Hospital the diarrhœa had increased in severity and he passed from six to seven stools daily containing blood and mucus. Latterly he had had pain in the abdomen, fever and sweating, but no rigors. During the last two weeks he had suffered from cough with expectoration, and he had vomited once after a fit of coughing. He had fallen off in weight considerably since the beginning of his illness.

Physical Examination.—Pale, thin and sallow; skin moist, tongue clean. Temp. 99° F., pulse 98, resp. 22. Thorax large, costal edge a little everted, respiration chiefly diaphragmatic. Percussion note clear over both lungs. On auscultation, a few piping râles over both lungs in front; behind, crackling râles at both bases. Apex-beat in the fifth intercostal space in the mammary line; heart-sounds clear. Abdomen soft and nowhere sensitive to pressure. Edge of liver palpable below the costal margin in the right mammary line: upper limit of hepatic dulness at the lower border of the fifth rib in the right mammary line, at the sixth rib in the mid-axillary line. Spleen enlarged: its edge palpable in the mid-axillary line. Urine: sp. gr. 1004, non-albuminous. Stools liquid, brownish-red, offensive, containing many particles of mucus and some blood. On microscopic examination, numerous moving amœbæ, blood corpuscles and pus cells.

Liquid diet.

Quinine injections, 1–2500, three times daily. Quin. sulph. 0.3 gm. t. i. d.

February 20th. Light yellow liquid stool, containing a small amount of mucus but no blood. No amœbæ found in this stool.

February 26th. Irregular fever since the day of admission. During the first three days the temperature ranged from 99°–100° F.; from the 18th to the 22d, 98.3°–102.8° F.; since the 23d, only once above 100° F. Pulse, 88–120; resp. 24–30. On the 18th

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there were seven stools; on the 19th, eight; from the 20th to the 24th, two to three; on the 25th, only one light yellow stool, formed and containing neither mucus or blood. On microscopic examination a few pus cells but no amœbæ were found.

March 3d. Quinine injections discontinued.

March 6th. One stool, formed and rather soft, without mucus or amœbæ.

March 31st. Four stools; a moderate number of amœbæ were found, some of which were active.

Quinine injections, 1–2500.

April 23d. Stool examined to-day was formed, and contained a few flakes of brownish mucus: no amœbæ were found.

May 14th. The patient's general condition was good; he was no longer anæmic, had gained 25 lbs. in weight, and his stools had been perfectly formed for more than a month and contained no mucus, blood or amœbæ. The spleen was still enlarged; the other viscera appeared to be normal.

He was discharged well.

CASE XIII.—*Dysentery of eight months' duration, beginning with an acute attack and becoming chronic. Amœbæ present in the stools, then disappearing for a few days. Reappearance of motionless and somewhat altered amœbæ in formed stools during convalescence. Improvement.*

Jacob K., aged 47, was admitted to the Johns Hopkins Hospital February 24th, 1891. He had lived in Maryland and, with the exception of an attack of malaria twenty-five years ago, had enjoyed good health up to the date of his present illness. About the end of June, 1890, he had severe diarrhœa, accompanied by much abdominal pain which was at first localised about the pit of the stomach, but subsequently over the bladder and in the left iliac region. The stools were very frequent and contained much mucus and blood. Toward the end of August the diarrhœa and abdominal pain moderated somewhat, but he still passed every morning two stools containing blood, followed during the day by two more natural stools. He had been sleepless at night and suffered pain when going to stool, for which he was in the habit of taking opium. During the autumn he had vomited occasionally. His appetite throughout had been capri-

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cious, and he had lost more than twenty pounds in weight. He felt weak and had some difficulty in urinating, but had never suffered from chills, sweating or fever.

Physical Examination.—General nutrition fairly good; slight anæmia; skin moist, complexion very sallow; tongue red, fissured and a little swollen. Temp. 100° F.; pulse 88, regular, tension increased; radial, brachial and temporal arteries thickened and tortuous; slight pulsation in the vessels of the neck. Thorax long, narrow and deep, slightly depressed in the lower sternal region; expansion equal on the two sides; slight hyper-resonance anteriorly over both lungs. On auscultation, the breath-sounds everywhere breezy, and expiration prolonged. Area of cardiac dulness diminished; aortic second sound accentuated and ringing, second sound reduplicated to the left of the sternum. Abdomen flat and soft; extreme sensitiveness to deep pressure between the navel and the pubis, but not in the flanks or over the liver; in the right and left iliac regions slight induration extending from the anterior superior spine two-thirds of the length of Poupart's ligament; this was more marked on the right than on the left side. The liver dulness extended in the right mammary line from the lower border of the sixth rib to the costal margin. The spleen was not enlarged.

Urine: sp. gr. 1012, slightly albuminous, without tube casts.

Liquid diet.

February 25th. Temp. 97.4°–98.8° F. Seven stools, small, very offensive, liquid, and containing blood, mucus and a small quantity of formed fæcal matter.

February 26th. Pain in the abdomen, which was very tender on palpation.

February 27th. Six stools containing numerous active amœbæ.

Quinine injections, 1–2500, and quin. sulph., 0.3 gm., three times daily.

March 3d. Two stools; no amœbæ found. Quinine injections discontinued.

March 5th. Three stools: one of these was formed, and adhering to it there was some clear mucus, in which a considerable number of amœbæ were found. The amœbæ were all motionless; the ectosarc distinct and very glassy in appearance, the endosarc containing highly

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refracting vacuoles and, in some of the amœbæ, appearing distinctly shrunken.

March 9th. Quinine injections, 1–2500.

March 23d. Since March 9th, from two to three movements of the bowels daily, following the quinine injections.

Quinine injections discontinued.

April 14th. Not more than one movement daily; frequently, none for two days. Fæces formed, of normal color, and containing very little mucus. A few amœbæ were found, which were motionless and somewhat altered as in the last examination.

The patient insisted on leaving the hospital, and was discharged much improved. When seen three months later he appeared perfectly well. He was then having one formed movement daily. No opportunity was afforded of examining his fæces.

CASE XIV.—

In addition to these cases we are indebted to Dr. R. W. Johnson for the clinical notes of the following case, which died at St. Joseph's Hospital in this city.

J. M., white, aet. 40, admitted to St. Joseph's Hospital September 22d, 1890. Said to be suffering from malarial or bilious dysentery. Had been having chills and sweating and general malaise for three weeks. His stools were frequent, not copious, bloody and very liquid. Temp. ranged from 101°–103° until two days before his death when it ran to 107° falling rapidly to 100°. Three days before death he passed a large amount of clotted blood. His liver was enlarged and painful. Abscess was diagnosticated and a small amount of creamy pus withdrawn by the aspirator.

Autopsy.—Made at St. Joseph's Hospital for Dr. Johnson; 36 hours after death. Acute croupous pneumonia of the left lung. Peritoneum smooth. Diaphragm on the right side at lower border of fourth rib. Liver 8 cm. below the ribs in line of nipple and 11 cm. below the ensiform cartilage. The liver is adherent to the diaphragm over a space 9 cm. in diameter. Just below this adhesion there is a furrow 1 cm. in depth extending downwards and backwards across the right lobe of the liver. There is an abscess in the right lobe of the liver just beneath the adhesion. The abscess cavity is 15 cm. in diameter and contains in the middle a thin

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bloody pus. Outside of this the pus is greyish but more transparent than ordinary pus. The abscess wall is covered with a layer of dark brownish necrotic material not stained with bile. In most places the line of the abscess is a sharp one, separated from the surrounding liver by a zone of connective tissue; but in places the line is broken and uneven, extending so deeply into the surrounding liver tissue as to form almost separate abscesses. The liver elsewhere is very pale. The gall bladder contains a small amount of clear thin yellowish bile.

The large intestine is contracted. There are numerous ulcers of various shapes and dimensions in the mucous surface. They are most numerous in the cæcum and ascending colon, the edges of the ulcers are undermined, the ulcers themselves round or oblong in shape with their long axes transverse to the intestine. They contain greyish gelatinous contents which can be lifted from the base of the ulcer and from beneath the overhanging edges. In the transverse colon the ulcers are larger. In one place the floor of the ulcer is formed by the longitudinal muscular tissue. In this ulcer there is an oval opening from which a small blood clot projects. Some of the smaller ulcers project above the general surface and communicate with one another by fistulous tracts in the submucous tissue. The stomach and small intestines are normal.

Microscopic examination of the contents of the ulcers showed numerous amœbæ. Most of them were not motile. In this condition they are generally round, contain numerous vacuoles and are strongly refractive. Several slowly moving amœbæ were found. In the contents of the liver abscess and in the wall there were numerous non-motile forms, but no moving ones were found.

CASE XV.—*Dysentery, beginning with an acute attack and lasting six weeks. Amœbæ not found during life. Peritonitis from perforation of the colon. Death. Autopsy.*

Geo. Ischekausky, aged 25, white, admitted to hospital September 11th, 1891, complaining of pain in the abdomen and diarrhœa. He was born in Poland and was never ill before coming to this country. He spoke no English, and from statements of his friends his illness began about four weeks before entering the hospital. He had been working on a farm in the county, and attributed his illness to eating green fruit. His first symptoms were pain in the stomach and head.

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The diarrhœa began four weeks ago; the stools have been liquid and he has not noticed any blood in them. For some time his bowels moved six to seven times a day, but latterly more frequently. At no time have the stools been painful. On entrance he was emaciated and anæmic. A few sibilant and sonorous râles were heard in the thorax. Heart normal. The liver is not palpable; the dulness begins at lower border of the sixth rib. Spleen not palpable. Abdomen slightly distended and uniformly tympanitic. Considerable gurgling and tenderness on pressure in both iliac regions. The temperature ranged from 97°–103.4°. On September 18th, had twenty-two stools, greenish-yellow in appearance and liquid. Some of them contained much blood. On 19th there was great abdominal pain. During the last few days most of the stools were passed involuntarily. The stools were examined for amœbæ, but none were found. The examinations of the stools was attended with much difficulty, for they were passed in the bed-linen, from which portions were scraped off and examined. He died September 21st, at 12 m.

Autopsy.—Anatomical diagnosis. Extensive ulceration and sloughing of large intestine. Peritonitis from perforation. Multiple abscesses of the liver. Catarrhal pneumonia. Body 158 cm. long, slightly emaciated; abdomen distended, slight hypostatic congestion. The mucous membranes in high degree anæmic. The subcutaneous fat very slight in amount; the muscles pale and soft. On opening the peritoneal cavity there is no escape of gas. The parietal peritoneum is cloudy, covered with a very slight fibrinous exudation, and there are a few small ecchymoses scattered over it. The intestines are covered with the adherent omentum. There is a slight exudation on this and between the loops of adherent intestines; considerable exudation can be seen through the omentum. On lifting the adherent omentum there is a free escape of gas from a cavity between the colon, the adherent omentum and coils of the small intestine. The walls of the cavity are covered with a dirty yellowish membrane. It contains a quantity of putrid pus mixed with fæcal matter. Outside of this cavity the coils of intestine are lightly adherent and there is some exudation best marked at the line of adhesion. The exudation is fibrinous, rather gelatinous and transparent. The mesenteric glands are very slightly enlarged. The liver is slightly adherent to the diaphragm by fresh adhesions. The diaphragm on right side

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at fourth intercostal space; on the left at the lower border of the fifth rib.

The lungs are slightly adherent by old adhesions. The pleural surfaces smooth save for these adhesions. Scattered through both lungs there are a few calcified nodules. In both lungs, but especially in the lower lobe of the right, there are a few very small foci of catarrhal pneumonia. The heart is normal.

Liver is large; size $24 \times 19 \times 8$ cm.; weight 1892 grammes. The capsule is smooth; the gall-duct open, and the gall-bladder contains a small amount of viscid, dark bile. It is of a rather pale brown color, and the lobules are only visible in places. Scattered over the surface there are numerous small white, opaque areas, not surrounded by a zone of hyperæmia, and not elevated above the surface. On section the liver is pale and opaque-looking; the lobules are not visible. There are numerous small white foci similar to those seen on the surface. They vary in size from 1–5 mm. On being cut across there is no escape of their contents. They have rather a dry appearance and the contents can be removed, leaving a ragged wall.

The spleen, kidneys, pancreas and adrenal glands are normal.

The stomach and small intestine contain semi-fluid, bile-colored material. The mucous membrane is hyperæmic in places.

The large intestine is generally adherent by fresh adhesions. The cavity mentioned between it and the adherent loops of small intestine communicates with the lumen by a ragged opening 1 cm. in diameter. The entire intestine is soft and tears easily on attempting to remove it from the body. Its walls are thickened and it is greatly dilated; the average circumference being 14 cm. It contains numerous ulcers and extensive sloughs from the cæcum to the anus. In the cæcum there are numerous round elevations often communicating with the lumen of the bowel by a small opening. Others apparently have no opening. There are also large, irregular ulcers, filled with a greyish-looking material, on removal of which the muscular coat is seen. In the transverse and descending colon, and in the rectum there are large, irregular ulcers, covered with dark sloughs which float up in water. On removal of these in some places the transverse muscular coat is laid bare, and in other places the ulceration extends through this to the longitudinal muscular coat or the peritoneum. All of these ulcers, both the large and small, are greatly undermined,

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and the base is rough and irregular with numerous points of ecchymosis. All the coats of the intestine are thickened and œdematous. There are also elevated ridges frequently leading off from the ulcers which contain on section the same whitish, semi-transparent material, as the contents of the abscesses. The lymph glands of the meso-colon very slightly enlarged and not softened.

Microscopic examination shows immense numbers of actively moving amœbæ in the general contents of the large intestine, in the peritoneal exudation, and in the material from the ulcers. They were present in the small abscesses of the liver, but not so numerous as in the intestine. Cover-slip preparations of the contents of these abscesses stained for bacteria gave negative results as to bacteria. No amœbæ were found in the lymph glands of the meso-colon or the mesentery.

Cultures were made at the autopsy from the peritoneal exudation; from the abscess cavity in the peritoneum; from the liver, spleen, kidneys, lungs and heart.

In the peritoneal cultures both the bacillus coli communis and staphylococci were found. The cultures from the liver and bile remained sterile. In the kidney the colon bacilli and staphylococci were found. The blood from the heart was sterile. Through a mistake no cultures were made from the liver abscesses.

General Clinical Review.

The cases given above in detail illustrate the form of dysentery which is associated with the presence of amœbæ in the evacuations and in the various anatomical lesions. A review of the disease as a whole, including a comparison with cases of catarrhal dysentery and of diphtheritic dysentery, will serve to further accentuate the specific character of the disease and differentiate it from other forms of intestinal disorder characterised by diarrhœal evacuations.

1. *Classification of Cases.*

A classification of "dysenteries" has frequently been attempted from an ætiological, an anatomical and a clinical point of view, or some combination of these. Recognising as we do a common ætiological factor and an identical anatomical process in all cases of this form of dysentery, we have adopted a purely clinical classification,

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which is simple and free from obscurity, and is based on the severity of the intestinal symptoms. It should be observed, however, that like all clinical groupings it is more or less arbitrary, and that cases will be observed which pass insensibly from one grade of severity into another; but in the main it will be found that cases may be ranged under one of the following divisions:—

1. Dysentery of moderate intensity.
2. Grave or gangrenous dysentery.
3. Chronic dysentery.

This is essentially the division proposed by Dutroulau.[50] The dysentery he described was the so-called tropical dysentery, which is identical with the one now under consideration. He insisted on the unity of dysentery as observed in the tropics, and drew attention to the fact that most of the authors who had written on dysentery occurring in temperate climates, had included in their classification forms of intestinal flux, occurring sporadically or epidemically, which differed clinically and anatomically from the dysentery of the tropics.

Kelsch and Kiener,[38] speaking of dysentery observed in tropical or sub-tropical countries, adopt a similar division, and allude to the distinction which must be drawn between dysentery and certain disorders of the bowels (*entérites*), which are liable to be mistaken for it, viz., acute and chronic intestinal catarrh, and a mycotic affection accompanied by necrosis (*entérite nécrosique parasitaire*).

Bérenger–Féraud [51] gives a partly clinical and partly anatomical classification, while among European and American authors (Virchow, Griesinger, Heubner, Baly, Woodward) there is a consensus of opinion to recognise a catarrhal and a diphtheritic form of dysentery. Whittaker,[51A] while adopting a provisional division into catarrhal and specific forms, remarks that a classification which does not indicate differences in ætiology is necessarily inadequate.

2. *Onset; Course and Duration; Termination.*

In each of these respects amœbic dysentery stands out in marked relief from other forms of intestinal flux. It is characterised by a variable mode of onset, an irregular course frequently marked by intermissions and exacerbations, an unusual latitude between its extreme periods of duration, and a tendency to chronicity.

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The *onset* is either abrupt or gradual. In the first case, without any premonition, the patient is seized with colicky pain in the abdomen and diarrhœa; he may or may not suffer from nausea and vomiting, and in the great majority of cases there is no fever. The stools are frequent, watery; they may or may not contain blood at first, but subsequently do contain both blood and mucus. When much blood and mucus are present from the first, the stools are small and not so liquid. In the second case, after a variable period of slight ill-health, a not very frequent and painless diarrhœa sets in, which alternates with short periods of constipation. Blood is not at all or only occasionally observed in the stools, which are watery and contain more or less mucus.

The *course* of the disease is marked by an irregularity in the abdominal symptoms which appears to be entirely independent of the mode of onset. At irregular intervals there are intermissions and exacerbations of the diarrhœa, for which no obvious cause can be assigned. These were observed, as may be seen by a reference to Cases I, IV, IX and X, particularly in chronic cases and in those of moderate severity; in the gangrenous form there were no actual intermissions, but a partial abatement followed by an exacerbation was of common occurrence. The relation which these intermissions and exacerbations bear to complications will be referred to further on. We have alluded to the tendency which there is in this disease to pass from one grade of severity into another. For example, a case of moderate severity may subside into a chronic dysentery, or prove fatal from an acute exacerbation and at the autopsy show the lesions of gangrenous dysentery. Or again, chronic dysentery may in its course present an acute exacerbation so severe as to be indistinguishable from a primary grave attack.

In our series of cases we have not had occasion to observe any true relapses. Further observations will show whether these are as frequent in amœbic dysentery in America as they are in the cases observed in the tropics.

The tendency to chronicity is sufficiently illustrated by the protracted course of the less grave forms and the large percentage of chronic cases in the series.

The *duration* of the disease is extremely variable. It may be stated generally that the more abrupt and severe the onset, the shorter

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was the duration of the disease. This is illustrated by the cases of gangrenous dysentery which we have observed, in which the duration in each case was 22, 34 and 16 days respectively. In the last case a diphtheritic inflammation of the ileum was present as well as dysenteric ulceration, and this undoubtedly hastened the fatal issue. The cases of moderate severity present a much wider range of duration, in in our series from six to thirteen weeks being the usual range. Occasionally the cases are even more protracted without presenting the features of chronic dysentery; in Case X, which ended fatally by abscess of the liver, the disease had lasted five months, but the lesions were not those of chronic dysentery. The duration of chronic cases varies between equally wide limits, but is to be measured by months and years rather than by days and weeks. A chronic state may be inferred when a moderate diarrhœa has continued for many weeks, with or without intermissions, and the stools are liquid, homogeneous, without blood and unaccompanied by pain.

The disease may *terminate* in recovery or death at any period of its course. Recovery is always gradual and may be interrupted by a short and temporary return of the diarrhœa with amœbæ in the stools (*vide* Cases XII and XIII). These slight recrudescences are analogous to the more serious exacerbations which form such a characteristic feature of the disease. Convalescence is uneventful but protracted, on account of the anæmia which is always present in a greater or less degree in the more protracted cases. A fatal issue may result from the severity of the intestinal ulceration, from exhaustion in prolonged cases, or from a complication (abscess of the liver, peritonitis from perforation, hæmorrhage, or the supervention of a diphtheritic inflammation of the intestine). Any of these modes of death may occur in each of the forms of dysentery, but death from abscess is more common in cases of moderate intensity and in chronic dysentery than in the grave form, in which the severity of the intestinal lesions or one of the other complications (peritonitis and diphtheritic enteritis) is a more usual cause of death.

3. *Analysis of Symptoms.*

(*a*) *General condition.*—A patient coming under observation in the course of a dysentery of moderate degree presents some such condition

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as the following: Muscular weakness, slight emaciation and blanching of the mucous membranes. Decubitus indifferent. Expression dull, sensorium clear. Skin often dry and inelastic, distinctly sallow. Tongue pale and flabby, moist and more or less furred. Abdomen of normal appearance, or more frequently retracted. Temperature not usually above 100° F., often normal. Pulse ranging from 70–90, and respirations from 18–30. Appetite impaired, and sleep disturbed by more or less frequent evacuations of the bowels.

A departure from or an accentuation of certain features in this clinical picture is observed in grave and in chronic dysentery. In the former the evidences of prostration are more marked; the face is drawn, slightly cyanosed or flushed, and the expression anxious; the mind is usually quite clear. There is almost complete anorexia, intense thirst and sleeplessness. The abdomen is greatly retracted, and there may be free sweating. The temperature is frequently normal or subnormal; the pulse small and rapid, and the respirations proportionately accelerated. In the latter, the progressive anæmia and loss of flesh are the special features which dominate the intestinal symptoms. The skin in chronic cases is dry, harsh, of a dull greyish-yellow color, and might be compared to dirty chamois-leather.

The *anæmia*, which is present in all cases of protracted dysentery, deserves special mention. The deficiency exists both in the corpuscular elements and in the hæmoglobin in about the same proportion. Further studies will show whether it differs materially from secondary anæmiæ due to other causes. It is, no doubt, to be attributed partly to direct loss of blood in small quantities from the intestinal mucous membrane, and to malnutrition. But this will hardly explain grave anæmia occurring in chronic cases in which appetite and digestion are fairly good, and no blood has been passed per rectum at any period of the illness. In such cases we must seek the cause in the amœbæ themselves, which, as we have observed, are active destroyers of the red blood corpuscles.

(*b*) *Diarrhœa* is the principal and in some cases the only feature of the disease. It is subject to great variation in character and frequency, both in the different grades of severity of the dysenteric ulceration and at different periods in the same case. We have already noted the occurrence of intermissions and exacerbations, which are the special characteristic of the diarrhœa, and may be observed at any

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period of the illness. The exacerbations often begin suddenly and subside in the same manner. They may last from one or two days to a week or ten days, and are progressively more severe and of longer duration in fatal cases. The intermissions have a wider range of duration, from one day to three weeks, and during this time the fæces are soft or even formed, but mucus is usually found adhering to them. We observed that these intermissions and exacerbations were most marked in cases complicated by abscess of the liver. The involvement of the liver did not coincide with any exacerbation of the intestinal symptoms: on the contrary, as may be seen by a reference to Cases II, IV, IX and X, there was at this time either a continuous diarrhœa of moderate severity, or slight looseness of the bowels alternating with constipation. The exacerbations occurred many days after there was evidence of hepatic and pulmonary involvement.

The *stools* are extremely variable in frequency, amount, and character, not only according to the severity of the intestinal ulceration, but also from day to day in individual cases.

In *gangrenous dysentery*, they may, at first, number thirty or forty in twenty-four hours, but subsequently they decline to from twelve to eight, and even to three or four toward the end of fatal cases. It may be that this is due to a gradual loss of expulsive power in the bowel. The amount voided at each movement of the bowels is at first small, and often consists entirely of clear or turbid masses of mucus, mixed with more or less bright blood and occasionally small fæcal masses. As ulceration advances the stools become more copious, watery, and less homogeneous: blood is less frequently observed, and small shreddy masses of a greyish or light yellow color appear, mixed with blood-stained mucus. When extensive sloughing is taking place the character of the stools is even more varied. They are of a greyish, greenish (resembling spinach, or the green scum seen in stagnant water), reddish-brown or variegated color, sometimes quite liquid, at other times pultaceous, and have a very penetrating offensive odor. Mixed with finely divided shreddy detritus, mucus, and streaks of dark blood, there are seen larger, tough, stringy masses of necrotic tissue of a greyish or yellowish-brown color. One of these measured 11 cm. in length. No characteristically purulent stools are passed, but the somewhat slimy, gray, liquid movements contain more

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pus cells than the others. In all the stools there are found more or less numerous small, opaque or translucent, gelatinous, greyish-yellow masses, 1–3 mm. in diameter.

In *dysentery of moderate severity* of abrupt onset the stools are, for a week or ten days, similar to those seen at the outset of gangrenous dysentery. If the onset has been gradual, liquid brownish-yellow stools containing mucus, streaks of blood, and many of the gelatinous greyish masses, are the rule. Of such movements there are from four to ten in twenty-four hours, and a flux of this degree, but more irregular and without blood, may continue for weeks. During an acute exacerbation the stools increase in frequency and resemble those of the second period of gangrenous dysentery; sloughs are occasionally found and blood usually reappears.

The stools of *chronic dysentery* have a more uniform and homogeneous appearance. They are watery or of the consistence and appearance of thin gruel, of an earthy or dull yellow color, and contain few or many particles of clear mucus. During an exacerbation blood and greenish pultaceous material may be seen; in intermissions of the diarrhœa, the fæces are soft and pasty, or even formed, but mucus is persistently present. Curds of milk and other undigested food are often seen, and the color of the stools may be altered by various drugs.

The reaction of dysenteric stools is generally alkaline, occasionally neutral or amphoteric, and more rarely acid. The relation which the reaction bears to the presence of amœbæ will be mentioned further on.

Microscopical Examination.—In the small, mucoid and bloody stools passed in the acute stage, red blood corpuscles, leucocytes, and large round or oval epithelioid cells are seen in varying proportions. The latter are often in groups of three or more; their protoplasm is granular; their outline sharp, and they show a distinct, somewhat eccentric, round or oval nucleus, about the size of a red blood corpuscle (Zeiss D). Many of them contain small fat drops, and some, large clear spaces. They may closely simulate amœbæ, from which they differ in being non-motile, and in refracting light less strongly. Cylindrical epithelium is not frequently seen. Bacteria are not numerous at this stage. The *cercomonas intestinalis* is sometimes present in considerable numbers.

At a later period, in gangrenous dysentery, and also in that of moderate severity, the cellular elements are less numerous, and float

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in a mixture of shreddy or granular detritus and bacteria. Elastic tissue fibres are observed, but the large or small necrotic-looking masses are devoid of any structural appearance.

Charcot's crystals, triple and earthy phosphates are very commonly seen.

In the liquid, greyish or yellowish-brown stools of chronic dysentery, recognisable elements are still fewer, and this makes the detection of amœbæ much easier.

Amœbæ are found in all varieties of dysenteric stools, and at all periods of the disease. A full description of them has already been given, and we shall now refer only to some features which have a special clinical interest.

They vary in number in different cases and at different periods in the same case in proportion to the severity of the intestinal ulceration. For example, they were much more numerous in severe acute cases, and in the exacerbations of cases of moderate severity, than during the periods of continuous but moderate diarrhœa. To this general statement an exception must be made in Case VIII, in which amœbæ were not found in the stools but only in sections of the ulcers. It is possible that the supervention of diphtheritic inflammation of the intestine may account for the absence of amœbæ. In the early bloody mucoid stools they are fairly numerous and evenly distributed. In the later and less homogeneous evacuations they are more numerous but less evenly distributed, being most abundant in the greyish-yellow gelatinous masses above referred to, next in the particles of clear or opaque mucus, and finally in the fluid portions of the stools; in the shreddy masses of detritus they are only occasionally found. In the homogeneous discharges of chronic dysentery they are, as a rule, less numerous than in acute cases, but are found in all portions of the stool. Even during an intermission of the diarrhœa, when the stools are soft or formed, amœbæ may be found in the particles of mucus adhering to the fæces (Case IX). We also observed that they varied considerably in number from day to day in stools of the same character. Amœbæ were not found in the stools of Case XV during life, though they were very numerous in the intestinal contents at the autopsy and must have been present. They were probably overlooked in consequence of the difficulty of making a proper examination.

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As recovery proceeds the amœbæ gradually disappear, but may be seen for a considerable time after the evacuations appear quite normal. A transient return of the amœbæ was noted during convalescence in Cases XII and XIII.

Variations in size and activity are frequent. Small amœbæ were particularly noted in Case VI, large and very active ones in Case X, and sluggish ones in Cases XI and XIII. Red blood corpuscles were always seen more frequently and in greater number in the more active amœbæ. It cannot yet be stated generally that the greater activity of the amœbæ corresponds to a more active ulcerative process in the bowel, but our observations, as far as they go, suggest such a conclusion.

The reaction of the stools has a very definite bearing on the presence and activity of the amœbæ. Cunningham [7] stated that the amœbæ are found only in alkaline or neutral, never in acid stools; and that they disappear from alkaline stools as soon as these become acid, the change occurring with a rapidity which is in inverse ratio to the degree of alkalinity (usually from ten minutes to an hour). In Case XI, observations specially directed to this point were made for six weeks. In the alkaline and neutral stools active amœbæ were constantly found in considerable numbers; in acid stools they were scarce and only occasionally motile.

The detection of actively moving amœbæ in dysenteric evacuations will be greatly facilitated if attention be paid to the following points:

1. The stools are to be passed into a warm bed-pan, and kept at a temperature of 30° to 35° C. until an examination is made.

2. The examination should be made as soon as possible, before the stools become acid.

3. The portions of the stools which have been noted as containing amœbæ in greatest abundance should be carefully looked for and examined.

A magnifying power of 400 diameters is the most suitable for the detection of the amœbæ, but they may be found even with a magnifying power of 100 diameters. For continued observation some form of warm stage is essential.

For purpoees of comparison a description of the stools in (1) catarrhal dysentery and (2) diphtheritic dysentery is appended.

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1. The stools of *catarrhal dysentery* are particularly characterised by their uniformity of character, quantity and frequency in all cases. With the abrupt onset there are evacuations consisting of bright blood and very viscid clear mucus, mixed with more or less fæcal matter. As early as the second day the stools may consist entirely of mucus and blood (especially if the patient be under strict regimen), of a faint blood-like odor, and so viscid that the bed-pan may be turned upside down in many cases without spilling its contents. This condition lasts one or two weeks, the mucus becoming gradually more opaque, of a greyish-white color, sometimes blood-stained, the blood progressively diminishing in quantity, and a little grey, green, or brown, pultaceous detritus, or fluid fæcal matter, appearing in the stools. From this time the quantity of blood and mucus diminishes gradually, small formed fæcal masses appear, and about the eighteenth to the twenty-fifth day fully formed normal fæces are passed. In the more prolonged cases wholly pultaceous, yellow-brown or greenish (spinach) evacuations may intervene between the bloody mucoid stools and the passage of formed fæces. No sloughs were observed in any of the discharges. The frequency of the stools is greatest at the outset and, with very few exceptions, decreases continuously, by two or three movements every second or third day until convalescence is established. In this respect particularly the evacuations of catarrhal dysentery differ from those of amœbic dysentery. According to the severity of the case the patient passes during the first few days of his illness from fifteen to forty stools. The quantity passed is in inverse proportion to the number of the evacuations, the bloody mucoid stools being small, the mixed or wholly pultaceous ones more bulky.

Microscopical Examination.—Red blood corpuscles predominate in the first bloody glairy stools. With these are associated a few leucocytes and cylindrical epithelial cells, and constantly many of the large round or oval epithelioid cells described in a former section. These occur singly or in groups, and are quite characteristic of the discharges. In the later stools fewer red blood corpuscles and more leucocytes are seen; and, in the pultaceous stools the cellular elements are comparatively scarce. Bacteria are not commonly seen in the early mucoid stools, but are present in the later more fluid or pultaceous evacuations. No amœbæ were ever found in any of these discharges.

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2. The stools of *diphtheritic dysentery* are in the main watery. They have not the uniformity of character according to the period of the illness, which has been noted as a special feature of the discharges of catarrhal dysentery, and resemble more closely the thin "meat-washings" evacuations seen in some cases of gangrenous dysentery. They are of an earthy-grey, greyish-green, or reddish-brown color, and usually very offensive. Mucus is not present in any considerable quantity, but small blood-stained particles of it are seen floating in the fluid portions of the stool. Bright blood may be present at the outset, but much more commonly there are minute dark-red clots or a general bloody discoloration of the discharges. Shreddy and finely divided material of a greyish or reddish-brown color is almost constantly present, but we did not at any time note the presence of distinct sloughs. The number of stools passed daily varies with the intensity of the diphtheritic process, but there is no regularity in the number of evacuations from day to day. They are usually very numerous at first; during the course of the illness, however, the daily average is from seven to fifteen stools. The quantity passed at each movement of the bowels is small.

Microscopical Examination.—The cellular elements are relatively few, those most constantly present being cylindrical epithelial cells, showing more or less fatty degeneration. Red blood corpuscles and leucocytes are observed especially in the stools which contain most mucus and blood. Fibrin constitutes a large portion of the unorganized debris, and in all the stools bacteria were present in great numbers. No amœbæ were found in any of the stools examined.

(c) *Other Abdominal Symptoms.*—Abdominal pain, sensitiveness to pressure, tenesmus, nausea and vomiting, and hiccough, are more or less frequently observed in the course of amœbic dysentery.

Of these symptoms *abdominal pain* is the most constant. It occurs most frequently in the early stage of gangrenous dysentery and dysentery of moderate severity with abrupt onset, and in acute exacerbations. With the subsidence of the acute diarrhœa the pain decreases in intensity, but, as it often disappears entirely as gangrene of the intestine progresses, this cannot have any prognostic value except in conjunction with other favorable symptoms. In chronic cases severe colic is not complained of, save during exacerbations, but there is

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often a dull aching or burning pain and a sensation of weight about the epigastrium. The pain is described by the patients as cramp-like, tearing, or sometimes burning. It usually precedes and accompanies a movement of the bowels, and when severe is general, but subsequently is localised, usually in the lower abdominal zone.

Sensitiveness to pressure is present in many cases. It is neither very acute nor general, as a rule, and is elicited most frequently by pressure along some part of the course of the large bowel.

Tenesmus. Great importance has been attached to this symptom, which is considered pathognomonic by Woodward, and no doubt is so of catarrhal dysentery and of most cases of diphtheritic dysentery. Our observations in the amœbic form of dysentery, however, coincide with those of Dutroulau, who noted the infrequency of tenesmus in an extended series of cases of tropical dysentery. In our series tenesmus was noted in four cases, and it is to be observed that three of these were the gangrenous form of dysentery, which again is in accordance with the remark of Dutroulau, that tenesmus occurs chiefly in grave cases in which there is extensive sloughing. A burning sensation in the rectum and at the anus during and after the passage of fæces, on the other hand, is very generally complained of.

Nausea and vomiting occur especially at the outset, but may recur at irregular intervals during the course of the illness. The ingestion of food and, in the cases complicated by abscess of the liver and lung, paroxysmal coughing, were the immediate cause of both.

Hiccough was present only in the terminal stages of gangrenous dysentery.

(*d*) *Fever, Rigors and Sweating.*—Elevation of the body temperature is not a prominent feature in this form of dysentery, which in this respect is similar to the other forms. It is nevertheless true that, if careful thermometric observations be made, an elevation of temperature above the normal point will be found at some period of the twenty-four hours daily throughout the course of the illness. Exceptions to this statement must be made in severe gangrenous dysentery, in which the temperature is sometimes normal or subnormal for twenty-four hours or more, and in chronic dysentery, which may be absolutely afebrile for days and weeks. This point was illustrated in Cases IX, XI and XIII, but even these patients had transient fever at irregular intervals during their illness.

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An exacerbation of the diarrhœa may be accompanied by a slight increase of the fever, but this is not at all constant. The development of hepatic and pulmonary abscess always produces an elevation of temperature.

The temperature curve is quite irregular; there may be alternations of continuous, remittent or intermittent pyrexia throughout the illness, and this occurs independently of any complication. As convalescence is established the fever declines, though slight irregular elevations of temperature are seen even when the diarrhœa has ceased. The average range in uncomplicated cases is from 99°–101° or 102° F., and the evening temperature is generally higher than the morning temperature, though there are many exceptions to this rule.

Rigors did not occur in any of the uncomplicated cases, and only one of the patients with hepatic abscess suffered from them.

Sweating is observed in all the abscess cases, and in gangrenous dysentery is often associated with a subnormal temperature and feeble circulation. In dysentery of moderate intensity it is seldom seen, and in chronic dysentery the skin is preternaturally dry.

(*e*) *Circulation and respiration* are affected to the same degree that they are in other diseases attended by moderate pyrexia. With the abrupt form of onset there is an acceleration of the pulse, which ranges from 80–90 or 100, and is full and regular. The average range throughout the illness in uncomplicated cases of moderate severity is from 80–90, in grave dysentery 100–110, while in chronic dysentery the pulse rate only occasionally exceeds the normal. With progressive exhaustion in fatal cases the pulse becomes feeble, compressible, and more rapid, ranging from 120–140. In Case VIII, however, the pulse though small, thready, and compressible, ranged between 88 and 100.

The respirations are accelerated pari passu with the pulse. The condition of the circulation and respiration in the cases complicated by hepato-pulmonary abscess is considered in the next section.

(*f*) The *urine* was slightly albuminous in all cases but one, and in one case a few hyaline casts were observed. There was no disturbance of the functions of the kidneys or bladder in the cases of chronic dysentery and in those of moderate intensity, but in the gangrenous cases there was retention, and the quantity withdrawn by catheter was much below the normal. Vesical tenesmus was not complained of.

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4. *Complications.*

In the series of cases which we have had under observation the following complications were found: abscess of the liver, three times; abscess of the liver and lung (hepato-pulmonary abscess), four times; peritonitis with perforation, once, without perforation, once; intestinal hæmorrhage, twice.

1. *Hepatic and hepato-pulmonary abscess.*—This is by far the most important complication in amœbic dysentery, not only from the frequency of its occurrence, but especially from the additional gravity which it lends to the disease.

It is quite unnecessary and superfluous, except from an historical point of view, to enter here into a discussion of the conflicting and often obscure theories, which have at various times prevailed, concerning the relation of hepatic abscess to dysentery. The recognition of the same ætiological factor in both cases has reduced the question to the consideration of hepatic involvement simply as an event, but a very important one, in the course of the disease, analogous to the infection of the liver in carcinoma of the stomach, or of various viscera in the course of a pulmonary tuberculosis.

We have been impressed with the frequency of this complication in the series of cases which we have recorded; out of fourteen cases no less than seven, that is 50 per cent., were complicated by abscess of the liver, or of the liver and lung, a far greater proportion than has been noted in regions in which this form of dysentery is much more prevalent. Whether this be a fortuitous occurrence or not, can only be determined by a more extended observation of the disease. However this may be, it is certain that this complication is far more frequent in amœbic dysentery than it is in catarrhal dysentery or in diphtheritic dysentery, in both of which it is a rare event.

Abscess of the liver may develop in a dysentery of any grade of severity and at any period of the illness. In our series it occurred four times in dysentery of moderate intensity, once in gangrenous dysentery, and twice in chronic dysentery. Making due allowance for the disparity in the number of cases of each grade of severity, it will be seen from this that gravity of the initial attack, far from favoring the development of liver abscess, is rather prejudicial to it,

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as the patient is more likely to die from the severity of the intestinal lesions before infection of the liver can occur.

The *time* at which hepatic involvement occurs varies between very wide limits. For example, in Case III it cannot have much exceeded two weeks, as the total duration of the illness was only three weeks; while in Case I the patient had suffered from chronic dysentery for at least two years before abscess developed. Between these two extremes, however, there is, in dysentery of moderate severity, a period between the fourth and the twelfth week, in which hepatic abscess more commonly develops. There are two circumstances which militate against the exact determination of the period at which this complication supervenes in any given case of dysentery; the first is the often indefinite onset of the intestinal affection, and the second the obscurity, in many cases, of the symptoms and physical signs which suggest abscess formation. It is probable that abscesses, contiguous to—and arising by direct infection from—some diseased portion of the bowel, are likely to develop earlier than those deeply-seated in the parenchyma of the liver, and especially near its diaphragmatic attachment.

The most common *site* of the abscesses in our series was on the convex surface of the right lobe close to the coronary ligament, and in every instance of this kind there was involvement of the lung. This was the condition in Cases II, IV, and X, and probably in Case IX, in which there was an hepato-pulmonary abscess from which the patient recovered. Dutroulau, in a series of ten cases of abscess, found four in which the lung was involved, and in three of these the abscess was evacuated through the bronchi. We have suggested elsewhere that infection in such cases takes place, not by the blood-vessels or lymphatics, but by way of the peritoneum, and that the amœbæ would in that event find a resting place about the coronary ligament. This is of extreme interest from a clinical point of view, and explains the early and frequent involvement of the lung resulting in an hepato-pulmonary abscess, which as we have just noted forms a large proportion of all the abscess cases.

Graphic descriptions of the *course* and *termination* of abscess of the liver in general have been given by Dutroulau, Kelsch and Kiener, and others, who have had a large experience with it in tropical regions. We shall confine ourselves to a general description of the course, and

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an analysis of the symptoms, of those cases in which there is involvement of the lung and early evacuation by expectoration.

In a number of cases of dysentery there is, after a variable period of time, evidence of involvement of the liver and subsequently of the right lung. This is often first indicated by pain in the right side of the chest, and the onset or exacerbation of fever, sometimes with rigors and vomiting, and usually with profuse sweating. The attitude of the patient suggests pleurisy, respiration is accelerated, and there is cough with expectoration, which is at first scanty and mucoid, and suddenly becomes profuse, bloody and purulent, after a violent paroxysm of coughing. From an examination of the abdomen there is not usually evidence of much enlargement of the liver, and in no case have we been able to detect fluctuation over the area of liver dulness. The physical signs of the involvement of the lung are at first always obscure and indefinite, and may remain so even after the patient has been spitting pus for weeks. This is due to the fact that the abscesses, both in the liver and in the lung, are deeply situated. Even repeated exploratory punctures with a long and large-sized aspirating needle are often negative, probably on account of the viscidity of the abscess contents. Examination of the sputa in these cases sooner or later reveals the presence of actively moving amœbæ. The general condition of the patient becomes much more serious with the development of hepato-pulmonary abscess. His appetite fails and his sleep is disturbed by frequent paroxysms of coughing. He emaciates and becomes weak, anæmic and sallow. Frequently the diarrhœa which was moderate or altogether in abeyance, becomes severe and continuous, and the patient after many weeks of suffering eventually dies, in spite of operative measures, worn out by continued suppuration, profuse sweating and diarrhœa. A favorable issue is indicated by a diminution of the cough and expectoration, the disappearance of the amœbæ from the sputa, subsidence of the fever and sweating, arrest of the diarrhœa, and a gradual improvement in the patient's general condition. Recovery is tedious and may be interrupted by a temporary return of the symptoms.

The *duration* of the fatal cases in our series (Cases II, IV and X) was respectively nine, ten and eight weeks. In Case IX the patient was still expectorating a small quantity of muco-pus ten months from the period of onset.

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Analysis of symptoms.—Pain was complained of at some period in all the cases. It is felt most severely from the outset until discharge of the abscess occurs, after which it subsides, recurring with less severity throughout the course of the illness, especially during paroxysms of cough. It is generally sharp, lancinating or tearing, but at times does not amount to more than a dull, heavy aching. The pain is referred to the right hypochondrium, the epigastrium, the lower ribs on the right side, the right scapula or the right shoulder, and may remain localised, but not unfrequently it radiates from any one of these situations over the right side of the chest and the abdomen. When very severe in the right side it interfered with respiration, and suggested (and probably was) pleuritic pain.

Fever was invariably present, as an exacerbation of an already slight pyrexia. The pyrexia is of an irregularly remittent or intermittent type, and presents no special or distinctive characters. It is simply a reproduction, on a slightly higher scale and with a wider range, of the fever observed in uncomplicated cases. There is usually an evening elevation and a morning remission, with an average difference of from 2° to 3° F. between them, but there may be a reversed curve. Case II, which was seen at the beginning of abscess formation, showed during the first four days a very slightly remittent, almost continued temperature, then a distinctly remittent, and finally an irregularly intermittent temperature. The temperature rarely exceeds 103° F., and much more frequently ranges between 100.5° and 102.5° F. When communication with a bronchus has been established, the fever declines somewhat and is even more irregular. Its range varies from day to day, being sometimes from 3° to 4° above the normal, at other times 1° or 2°, and that only during a portion of the twenty-four hours. In Case IX, after the tenth day, the temperature rarely exceeded 99° F., and was frequently normal, notwithstanding the fact that the patient was expectorating mucopus freely for several months.

Rigors and Sweating.—Definite rigors were observed only in one case, but slight chilly sensations were frequently complained of. Profuse drenching sweats are very common, and occur irregularly throughout the course of the illness, during daytime or at night independently of the temperature.

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Nausea and Vomiting may be present at the outset just as in other febrile affections, but they more commonly accompany and follow a severe paroxysm of coughing.

Jaundice was observed in one case, but it was very slight, and difficult to distinguish from the general sallowness except in the conjunctivæ.

Circulation.—In all cases there is at first a moderate acceleration of the pulse, which ranges from 80 to 100, and is full, regular and of good volume. This condition is maintained so long as the patient is holding his own against the disease, but with increasing emaciation and loss of strength, the pulse becomes feeble and may range from 120–140. Dicrotism was not noted in any case. It may be said generally that, as in uncomplicated cases, the pulse varies more according to the patient's general condition than in relation to the temperature. With progressive failure of the circulation there is sometimes slight lividity of the lips and finger-tips.

Respiration is also accelerated, ranging from 24 to 32 or even 40. At first, especially before the abscess has found vent, there is dyspnœa, which appears to depend more on the severity of the accompanying pain than on the extent of lung involved, for when pain ceases there is little or no dyspnœa, notwithstanding the increased destruction of lung tissue.

Cough and Expectoration are early symptoms. The cough is at first short, hacking, painful, and repeated at frequent intervals, like that of pleurisy. It is often ineffectual, but may be followed by the expectoration of a little viscid clear mucus. In a few days the sputa became more abundant, slightly muco-purulent, and from time to time streaked with blood, or rusty. Microscopical examination shows principally leucocytes, red blood corpuscles and alveolar epithelium.

The patient now begins to suffer from exhausting paroxysms of cough, which are often started by a change of position, and finally, during one of these paroxysms, expectorates suddenly a large quantity of brick-red or brownish muco-pus, mixed with more or less pure blood. From this time, throughout the illness, a similar material is daily expectorated in varying quantities, usually after paroxysmal coughing.

The *sputa* deserve special attention as they have been considered to be almost pathognomonic of hepatic abscess. "When this hap-

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pens [evacuation through a bronchus], it is marked by the sudden expectoration of a dirty-red or brownish puriform matter. The peculiar color arises from the pus in its passage through the lung becoming mixed with blood and broken-down pulmonary tissue. There is no matter like it expectorated in any disease of the lung itself, and I believe that its appearance is pathognomonic of abscess of the liver, or at least of abscess perforating the lung. I have been led by it to detect an abscess of the liver of which I had previously no suspicion" (Budd). Morehead[46] has an observation to the same effect.

The expectoration is partly diffluent, partly tenacious, and partly frothy, its viscidity being proportional to the amount of mucus present. It is at first often bright red owing to a large admixture of blood, and subsequently of a dull brick-red, brownish-red, or rusty brown color and occasionally bile-stained. Intimately mixed with the muco-purulent mass are more or less numerous small yellowish-white fragments of a friable or cheesy consistency. After exposure to air for some time, the whole mass liquefies into a thin syrupy or oily homogeneous fluid, which has frequently been compared to anchovy sauce. Such a fluid was aspirated from an abscess of the liver which had not perforated the lung. The sputa are alkaline in reaction, and have a faint sweetish odor. In no case did we notice a putrid odor. At a later period they become more purulent, somewhat nummular, reddish-yellow, and contain less blood. If the patient progress toward recovery, the expectoration becomes more fluid and frothy, and contains less pus which is slightly blood-stained. On settling it separates into an upper frothy layer, a middle layer of slightly turbid fluid, and a thin bottom layer of muco-pus. The quantity expectorated daily varies from 25 cc. to 400 or 500 cc. Even more than this may be coughed up at the time the abscess first discharges itself.

Microscopical Characters.—The cellular elements observed are red blood corpuscles, leucocytes, round alveolar epithelial cells, oral epithelium, and irregular polyhedral fatty degenerated elements, which look like liver cells but do not show a nucleus. In the early sputa red blood corpuscles, and in the later sputa leucocytes, predominate, while swollen and degenerated alveolar epithelium is present in about the same proportion in all the sputa. Elastic tissue fibres from the lung, single or in groups, showing the characteristic wavy or curled appearance, are frequently found, more especially in the

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later stages. Orange-red rhombic crystals of hæmatoidin, needle-shaped crystals of tyrosin, single or in fan-shaped groups, of the same color, and Charcot's crystals are occasionally seen, with various bacteria and particles of food. The small cheesy fragments consist entirely of an amorphous granular material and oil globules.

Amœbæ are constantly present. They occur indifferently in all portions of the expectoration more or less abundantly, but on the whole they are not so plentiful as in the stools. The number observed from day to day is very variable. They vary in size and activity, but are generally larger than those seen in the stools, and very frequently contain red blood corpuscles. In Case X they were particularly abundant, and in Case IX scarce, disappearing as the cough and expectoration diminished. A general description of them has already been given. For their detection the same precautions should be taken as in the case of the stools, *i. e.*, the sputa should be kept at a temperature of 30°–35° C., and examined as soon as possible.

The physical signs of hepato-pulmonary abscess develop slowly, and at the onset are quite obscure and inconclusive.

Inspection of the thorax at this time usually reveals nothing abnormal. There may be less expansion in the lower thoracic zone on the right side than on the left, but, as the respiratory movements are very limited by reason of the sharp pain experienced during inspiration, the difference is barely appreciable. There was no bulging of the lower part of the thorax or partial obliteration of the right intercostal spaces in three cases; and in Case X it was observed only toward the close of the illness. The absence of these signs is probably due to the depth at which the abscess is situated, and to early evacuation through the bronchi, which prevents a very large accumulation of pus.

Palpation. In many cases the edge of the liver is palpable below the costal margin in the mammary line. This was the case in Cases IV, IX and X. Tenderness on pressure was present over the liver in Cases II, IX and X, and below the margin of the ribs in Cases IV and IX. In the latter instance it is often difficult to decide whether the sensitiveness is distinct from that over the adjacent colon. Tactile fremitus was diminished or quite absent at the extreme base of the right lung, and slightly exaggerated immediately above this point (indicating an encroachment of the liver upon the normal area

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of lung resonance, and a limited area of consolidation in the lower lobe of the lung—it was demonstrated by puncture with an hypodermic syringe that these signs were not those of pleural effusion). Fluctuation was not detected in any case.

Percussion demonstrated a moderate and progressive extension of the area of liver dulness both upward and downward. The former is the first to become evident and the more marked throughout. This was illustrated in Case II, which was seen in its incipiency and in which there was at first no downward extension of the liver dulness. The dulness may extend from two to eight centimetres or more below the margin of the ribs; the upper limit may reach the fifth rib, or later even the fourth in the mammary line; and behind it may extend to a short distance above the angle of the scapula. Immediately above this upper limit of dulness is an ill-defined zone extending round the right side of the chest, over which there is high-pitched resonance, sometimes of a tympanitic quality (Case II), and which shades off gradually into normal resonance: this zone is of greater extent behind and in the posterior axillary space than in front.

Auscultation reveals at first, over the zone above mentioned, an enfeebled respiratory murmur, which is faintly bronchial in character posteriorly, and is accompanied during inspiration by muffled crackling râles, most numerous posteriorly. At the extreme base of the lung breathing is barely audible or entirely absent. A friction rub is frequently heard below the right nipple and in the lower axillary space, and may be detected by palpation. The voice sounds are slightly diminished in intensity and may have a nasal quality. Over the upper half of the lung respiration is feeble as compared with the opposite side, but not altered in quality.

When discharge of the abscess has taken place through the bronchi, the physical signs are materially altered, particularly on auscultation.

On inspection no change is observed from the condition noted above. Palpation may show a recession of the edge of the liver towards the costal margin, and percussion a corresponding diminution of the area of liver dulness, chiefly in front and in the axillary space (Case II).

On auscultation, over the zone of high-pitched resonance, there is a much more pronounced tubular breathing, especially at the angle of the scapula and about the posterior axillary fold, and with inspiration there are numerous coarse bubbling râles, which are resonant

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or even gurgling when the patient coughs. The voice sounds are increased in intensity, articulate and nasal; the whispering voice may be distinctly transmitted (Case X) if the cavity be large and near the surface.

If recovery takes place these physical signs slowly disappear—the liver dulness gradually resumes its normal area, the lung becomes resonant throughout, the bronchial breathing becomes fainter or is entirely replaced by a slightly enfeebled vesicular murmur, and the rales become smaller, less resonant, and finally disappear.

2. *Peritonitis.*—Perforative peritonitis occurred in Case VI. There was no evidence of it during life from an examination of the abdomen, but the sudden collapse of the patient suggested the diagnosis which was confirmed by post-mortem examination. Perforation is more likely to occur in severe gangrenous dysentery than in dysentery of moderate severity or chronic dysentery, in which there is more or less of a reparative process at the floor of the ulcers.

Peritonitis without perforation was demonstrated post-mortem in Case X. The patient suffered severe abdominal pain for several days, and his abdomen was very sensitive to pressure, but not distended. In this case there was no sudden collapse.

3. *Hæmorrhage* from the bowel was observed in Case VIII, on the last day of the illness. 125 cc. of clotted blood were passed per rectum, and the patient, who was already very weak, died collapsed three hours later. It was also present in Case III and in Dr. Johnson's case.

Of other complications and sequelæ, viz., pneumonia, rheumatic joint affections, thromboses, œdemas and paralyses, we have had no experience in our series.

5. *Diagnosis.*

This is essentially the detection of actively moving amœbæ in the stools, in aspirated contents of hepatic abscess, or in the sputa. In most instances of amœbic dysentery a single examination of the stools affords positive evidence, in a few, repeated examinations may be required. This is particularly the case when the stools are partly formed, few in number, and contain only a little mucus, in which nevertheless amœbæ may be found by a thorough examination. In some cases complicated by hepato-pulmonary abscess the diagnosis

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may be first established by finding amœbæ in the sputa; the disease being at the time more active in the liver and lung than in the intestine (Cases II, IV and IX). Here the amœbæ have a doubly diagnostic value—they indicate hepatic abscess, and direct attention to intestinal ulceration, no suspicion of which has been aroused by the appearance of the fæces.

The diagnosis of abscess before its discharge may or may not be difficult from the history of the case and the physical examination. In all such cases we have invariably resorted to early exploratory puncture with an aspirating needle. When the abscess is centrally situated in the right lobe and of some magnitude, aspiration with a long needle generally gives positive results. (This was the case in three abscesses of apparently non-dysenteric origin, in which pus was obtained at the first aspiration of the liver). But when it is situated on the convex surface of the liver at the point of attachment to the diaphragm, and early invades the lung, extending rather more upward than downward; in such cases aspiration generally fails to reveal pus (Cases II and IV). As remarked before the viscidity of the abscess contents may be so great as to prevent their passage through the needle.

The diagnosis of small multiple abscesses and of superficial abscesses occupying the concave surface of the liver is more or less conjectural.

We have examined, in the course of our investigations, the evacuations in various diseases accompanied by some form of intestinal flux, viz., catarrhal dysentery, twelve cases; diphtheritic dysentery, three cases (verified by autopsy); membranous colitis, one case; late diarrhœa of typhoid fever, one case; chronic diarrhœa, five cases. In none of these were any amœbæ found. It is certainly important, from the point of view of diagnosis, prognosis and treatment, that the stools in all cases of prolonged diarrhœa for which there is no obvious cause, should be carefully examined for amœbæ.

We have also examined the pus from four liver abscesses, in cases in which there was no history of dysentery, and have not succeeded in finding any amœbæ. Three of the cases recovered after operation; in the fourth, which was fatal, a single, small, shallow ulcer was found in the rectum. Whether the three patients who recovered were suffering from latent dysentery or not, must remain a matter of conjecture.

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While insisting on the diagnostic value of the amœba in the stools and sputa, we do not underestimate the evidence derived from the history and course of the illness and from the physical examination of the patient. In any given case—a history of irregular and often intermitting diarrhœa, beginning gradually, or abruptly with bloody mucoid stools, and accompanied by slight fever, emaciation, loss of strength, and sallowness; particularly (in some cases), symptoms and physical signs pointing more or less clearly to hepatic and pulmonary mischief, are collectively strong presumptive evidence of this form of dysentery, and should suggest a daily thorough microscopic examination of the stools, if this has not already been done.

6. *Prognosis.*

Whether gangrenous, of moderate severity or chronic, amœbic dysentery is a serious disease, and its prognosis is always uncertain. Dutroulau pointed out that the gravity of this disease lies in its persistency, and was impressed by the observation that most cases which become grave do so by progressive exacerbations. The frequency with which even mild cases are complicated by abscess, and the constant tendency to chronicity in cases which are not rapidly fatal are the factors which make it impossible to estimate the probabilities at the outset of the illness. This is well illustrated by the large proportion of deaths which occurred in our series in cases of moderate severity. In uncomplicated cases a favorable issue may be expected when the fæces become formed and no longer contain amœbæ after repeated examinations.

7. *Treatment.*

A discussion of therapeutic measures based on a small number of observations is naturally inconclusive, and we may be excused for not insisting on the value of the considerations subjoined. A statement of the measures adopted and of the reasons which appeared to justify their employment is all that is necessary and advisable.

1. *General, Dietetic, and Symptomatic.*—In each of these respects the measures we resorted to were in no wise materially different from those generally recommended in similar cases.

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The patient was confined to bed and given a suitable amount of food in an exclusively liquid form throughout the illness until convalescence was well established. Milk was given, if the stomach tolerated it and there was no evidence from the stools that it was imperfectly digested. If not, egg-albumen, with occasionally some clear soup, was substituted for it. During convalescence the dietary was increased gradually until the patient was able to take a full diet without any resulting inconvenience.

Insomnia, nausea and vomiting were relieved in the usual manner.

Abdominal pain and tenesmus were alleviated by various local sedative applications, or if necessary by suppositories and subcutaneous injections of morphia, or enemata of starch and laudanum.

The distressing paroxysmal cough accompanying the development of liver abscess was best combated by the use of codeia, or if this failed, morphia in small doses.

Ipecacuanha was administered in Case VIII, after the manner recommended by surgeons in India, and in Cases IV, IX, X, XI, XII and XIII quinine was given by mouth, on theoretical grounds rather than from empirical evidence as to its value.

Iron was given during convalescence.

2. *Local.*—(*a*) *Sedative rectal injections* were employed, particularly in the gangrenous cases, when pain was severe and the patient was distressed by very frequent evacuations.

(*b*) *Parasiticide rectal injections.*—Solutions of bichloride of mercury of a strength of 1–5000 or 1–3000 were employed in Cases III and V. Half a litre to one litre of this solution was injected twice or three times daily, and each injection was retained from ten to fifteen minutes.

Injections of a solution of quinine of varying strength, 1–5000, 1–2500, and 1–1000 were used in Cases VII, X, XI, XII and XIII. The use of these injections was revived by Lösch, who found that a solution of 1–5000 killed the amœbæ. In Cases VII, XII and XIII, the amœbæ disappeared from the stools after a more or less prolonged use of the injections; in the two latter cases they returned in small numbers but generally inactive, when the injections were discontinued; in Case XII they disappeared after a renewal of the injections, and were not found in the stools a month later; while in Case XIII there were still a few inactive amœbæ in the stools

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when the patient left the hospital. In Case X, which was fatal, the injections were discontinued on account of the patient's hopeless condition. Case XI is still under observation, and amœbæ are quite as numerous as they were when the treatment was first instituted.

Of these solutions, from half a litre to one litre is injected three times daily. The patient is directed to retain the injection fifteen minutes, and gentle friction of the abdomen along the course of the colon is practiced during this interval. The passage of the enema is facilitated by placing the patient in the knee and chest position, and if the rectum is very irritable a cocaine suppository may be used a few minutes before the injection is given.

The results achieved are not brilliant, but warrant a more extended trial. This much can be said, that quinine injections do destroy amœbæ in the contents of the bowel, but whether they reach and destroy the amœbæ in the tissues is an open question. It is probable that the injections will be serviceable chiefly in incipient cases, and those in which the disease is limited to the descending colon, sigmoid flexure and rectum.

3. *Treatment of Complications.*—The question of operative interference arises when hepatic or hepato-pulmonary abscess has been diagnosticated. In any individual case, the surgeon should decide whether or not an operation is advisable, and at what point the abscess may be opened with the best chance of success. Abscesses involving the lung are certainly more difficult to reach and drain thoroughly than are those situated centrally in the right lobe, and the chances for the patient are correspondingly diminished (Cases II and IV). It is possible that abscesses due to amœbæ are less favorable for operation than those in which no amœbæ are found.

Note.—An opinion on the sources of infection must be purely speculative until the morphology of the pathogenic amœba has been fully worked out, and its habitat discovered. It seems probable that drinking-water is one, if not the chief, source of infection.

8. *The Intestinal Lesions.*

When we review the gross appearances which the colon presents in all these cases, we see that, although the lesions vary in character in the different cases and even in the same intestine, there are certain

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features which are met with in all cases. The most striking characteristic in all, and the one which first attracted our attention to this as a special anatomical form of dysentery, was the great thickening of the intestine. This was present in every case, but was much more marked in some than in others. Sometimes the thickening involved all the coats, but in some cases it was confined to the submucous coat; and in every instance it was more marked in this than in the others. There was not only a general thickening of the coat, due to an œdematous condition, but there were sharply circumscribed projecting nodular thickenings in which were small cavities filled with gelatinous-looking pus. These cavities communicated with the surface of the mucous membrane by a small opening. There were also sinuous tracts, sometimes representing an extension of the cavity, sometimes communicating with neighboring cavities, all filled with the same viscid gelatinous material as the cavities.

The elevated nodules varied considerably in size, and the openings were frequently no larger than the head of a small pin, or so large that the cavity was freely exposed; but even then the surrounding mucous membrane was deeply undermined, and there were often found sinuses in the submucosa leading off from these ulcers. These ulcers were filled with the same glairy gelatinous-looking material as the cavities; this could be lifted out, leaving the ulcer clean, sometimes portions of it still adhered to the walls. Examined fresh under the microscope, it nearly always contained numbers of the amœbæ, very large round swollen cells, red corpuscles and pus cells. These large round cells have a single nucleus; they are sometimes simply granular, at others, filled with fat drops. They are probably enlarged connective tissue corpuscles which have become free by the softening of the tissue around them. The whole material was so tenacious that it was difficult to press down the cover-slip upon it. The large cells in it may be mistaken for amœbæ, and indeed bear some resemblance to the resting form of these. At the autopsies moving amœbæ were found in the material from the ulcers, or in the intestinal contents, in all but two cases.

Most of the autopsies were made a very short time after death. In Case II the tissues were not examined until the following day, the autopsy being made late at night; and in Case XIV, thirty-six hours had elapsed before the autopsy could be made. In the first

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mentioned case numbers of actively moving amœbæ were found; but in the second a prolonged search showed a few only which were slowly moving.

The undermined ulcers in connection with the formation of cavities and sinuous tracts in the submucosa can be regarded as the form most frequently met with. They are found in the same intestine in connection with other forms of ulcers; and in some cases are much more prominent than in others. They are more generally met with in the last portion of the transverse and descending colon. There are other ulcers which are quite small with somewhat undermined edges, and which represent simply excavations in the thickened submucosa without distinct cavity formation in the tissue. In all of the cases, more particularly in Case X, there were ulcers which apparently represent a later stage of the excavated ulcers. In these the undermined edges had sloughed away, leaving the edges smooth in places; in others, only slightly undermined. They had a firm base which generally extended to the muscular coat. The extent of the sloughing varied. In Case VI, sloughs were met with as large as the palm of the hand. These sloughs were formed of the muscular coat which had been dissected up by the advance of the infiltration in the intermuscular tissue, in the same way that the mucous membrane was undermined by the submucous infiltration.

In every case the submucous coat was the most affected. This was infiltrated and œdematous, not only in the neighborhood of the ulcers, but in places which were free from ulceration. The ulcers certainly increased in extent by the gradual infiltration and softening of this tissue with subsequent necrosis of the tissue immediately over it. The roof which covers in the more or less closed ulcer is broken through, the infiltration and the softening continue at the sides, and an ulcer with deeply undermined ragged edges is formed. The base of these ulcers is clean, and is formed by the muscular coat. Although the ulcer advances in the submucosa by the gradual softening and disintegration of the tissue, the muscular coat offers a barrier to this molecular destruction. The upper layers of this may become necrotic; the cellular infiltration extends into it along the connective tissue septa between the bundles of muscular fibres, but it holds together. The infiltration gradually extends through it into the intermuscular connective tissue septa and the subserous coat, and

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then the same process which took place in the submucous coat is repeated here. The circular muscular coat is dissected up, the vessels supplying it are destroyed, and it undergoes necrosis as a whole. The large sloughs of tissue, which are cast off and frequently passed in the stools, are composed either of this coat or of the muscularis mucosæ. While these processes are taking place, the peritoneum becomes enormously thickened, and the same process of destruction is continued in this. There are adhesions formed with the surrounding viscera, and the greatly swollen intestine becomes convoluted and adherent to itself and to the surrounding viscera.

In the account which Annesly [42] has given of the disease in India, the deformities produced in the abdominal viscera by these adhesions are described at great length, and form a large part of his description of the pathology of the disease.

The mucous membrane seems to suffer least, or at any rate the lesions in it are for the most part secondary to those of the submucosa. The process is essentially one of advancing infiltration and softening in the submucous and intermuscular tissue, with following necrosis of the over-lying tissue. Plate I and Plate II, Fig. 1, show some of the forms of these ulcers. The characteristic undermining of the tissue is shown in Figs. 1, 2, 3, 4 and 7, Plate I. The cavity in Fig. 3 is a cross section of one of the sinuous passages in the submucous tissue. Fig. 4 gives a good representation of the great extent of the undermining of the mucous membrane; portions of it, like mushrooms, are connected with the tissue beneath by very small pedicles. The cavities beneath the mucous membrane were filled with the contents which have been described, but which have dropped out in the hardening and mounting. Fig. 5 shows an ulcer with smooth walls and base, extending down to the muscular coat, which in one place is broken through, and the infiltration, extends below this in the peritoneum, which at this point is also thickened and œdematous. The large sloughs spoken of, are formed in this way. Fig. 6 shows a portion of a large ulcer extending to the muscular coat. A portion of this coat has been destroyed, the ulcer at this point extending to the connective tissue.

It is possible to divide the ulcers into four forms: First, ulcers characterised by purulent infiltration, softening and cavity formation in the mucosa; these have a small opening on the mucous membrane,

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and often communicate with neighboring ulcers by passages in the submucosa. Second, ulcers with slight undermining of the edges, representing simply excavations in the thickened submucous tissue. Third, ulcers with smooth sides and clean bases. Fourth, ulcers with extensive sloughs adhering to the sides. Although these various forms at first sight seem rather different, the differences merely represent different results of the same process. They are due to the nature of the tissues; the arrangement in layers separated by rather loose connective tissue; the rapidity of the extension of the process, and probably to the combined action of other organisms with the amœbæ. For microscopic examination, portions of the tissue were hardened in alcohol, Müller's fluid, chromic acid and Flemming's solution. The best results were obtained after hardening the spread-out intestine in strong alcohol, and staining the sections in Loeffler's methylene-blue.

In all of the cases there was very little change in the mucous membrane at a distance from the ulcers. The layer of epithelium on the surface was generally absent, and here and there were slight erosions of the upper part of the glands. Most of these were found where the autopsies were not so fresh and the tissues not in such good condition. Lower down in the mucous membrane some of the glands were dilated and filled with accumulations of mucus and pus cells. In all of these dilated glands the epithelial cells were filled with mucus, and there seemed to be an increased amount of mucus in the epithelial cells generally. There did not seem to be any general round-cell infiltration in the mucous membrane, and no infiltration with leucocytes except in the immediate vicinity of the small glandular cysts which contained them. These changes in the mucous membrane were best seen in sections made parallel to it. In some of these sections cysts of considerable size with irregular walls were found, which were probably formed by the confluence of neighboring glands. There was also some proliferation of the lining epithelium, shown by numerous nuclear figures. Amœbæ were found in the mucous membrane in two situations. In a few instances they were found in the cystic dilatation of the glands at quite a distance from the ulcers. Some cysts were found with quite a number of amœbæ in them. In these cysts there was generally an absence of pus cells, but some contained both the amœbæ and pus cells. In the cysts

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with the amœbæ there was some accumulation of mucus. In one such place, represented in the plate, Fig. 3, Plate III, the epithelium has disappeared over quite a large surface of the cyst, and the wall is broken and imperfect. There are also amœbæ in the interglandular tissue adjoining the cyst. More frequently, however, the amœbæ are in the lower part of the mucous membrane, between and sometimes in the glands; and in this situation they evidently had come from the submucous tissue through the muscularis mucosæ. Fig. 1, Plate III shows such a place. There are numerous amœbæ in the submucosa, both in the lymphatics and in the tissues. They have entered the muscularis mucosæ which is swollen and œdematous at this place, and have extended into the mucous membrane.

In none of the cases was there any fibrinous exudation in the upper portion or on the surface of the mucous membrane. At the edges of the ulcers very interesting changes were found in the mucous membrane. The blood vessels were dilated and contained numbers of leucocytes. These were also found in considerable numbers in the tissue. Near the margin of some of the ulcers, the interglandular tissue was softened, the glands lying in a granular material containing numerous leucocytes and some granulation cells. The glands themselves were broken up, and only here and there portions of them represented by a few adhering epithelial cells were found in the granular detritus. This condition was not the one generally found; and in the same ulcer a portion would show the disintegration of the mucous membrane on one side of the ulcer only. More generally, and especially was this the case in the ulcers with smooth walls and bases, there was an enormous proliferation of the glandular epithelium. This was shown not only in branching and budding of the glands at the edge (Plate II, Fig. 4), but in a regular epithelial investment of the entire edge of the ulcer, which extended down over the undermined portion, and in some cases partly over the floor of the ulcer. In this new growth of the epithelium there were bud-like projections into the tissue beneath, and in some cases these had a lumen and represented newly-formed glands. In the cases characterized by the formation of cavities in the submucous tissue, the roof of the cavity was frequently entirely covered by a new formation of epithelium which had grown downward from the mucous membrane. Plate II, Fig. 3 shows a section which passes through one of these

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cavities at quite a distance from the opening on the surface. The roof is covered with a single layer of epithelial cells, which partially extends downward over the sides. As this epithelial growth proceeds downward, the cells first become cuboidal, and afterwards, almost flattened.

In most of the ulcers there was not only this proliferation of the epithelium, but the entire mucous surface was folded and turned in over the ulcer. This is seen in Fig. 4, Plate II; but is much more pronounced in Fig. 1, Plate IV. The entire mucous membrane with the muscularis mucosae is turned in, almost covering the ulcer. Very many nuclear figures indicating cell proliferation were found in the epithelium at all these points. They were best shown in the specimens hardened in alcohol and stained with Weigert's fibrin stain. Plate IV, Fig. 1 shows a section passing through one of the cavities in the submucosa. In this the contents were almost entirely pus cells which lay in a granular detritus mass. There were also in the mass a few amœbæ, some lymphoid cells, and some large swollen cells. There was a tolerably well-defined wall to the cavity, composed of very large mononuclear cells lying in a loose tissue. Between these large cells there were lymphoid cells in large numbers and pus cells. Between the central purulent mass of the cavity and the wall there was a space which contained many of the large cells. These cells were granular, the body of the cell stained with eosin, and they contained one, and sometimes two or three nuclei. Sometimes the nucleus did not stain. These large cells may easily be mistaken for amœbæ. They are evidently of the same character as the cells lining the cavity, and they have been separated from the wall by the gradual softening of the intercellular substance. These are the large swollen cells found in the fæces. There are also some amœbæ in this space along with the large cells.

The submucous tissue around the ulcer was infiltrated with cells. These were in part scattered diffusely through the tissue, and in part arranged in masses and rows. The connective tissue cells are large, pale and swollen, and in some cases their nuclei do not stain. The processes come out clean and distinct in the homogeneous-looking intercellular tissue. This tissue has lost its fibrillation; it is not arranged in bands, but is clear and homogeneous-looking. The cellular infiltration is most intense in the immediate neighborhood

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of the ulcer, but there are often seen masses of pus and round cells separated from the wall and close beneath the muscularis mucosae. At Fig. 1, Plate I, such a mass is seen immediately beneath the muscularis mucosae, which at this point, together with the lower layer of the mucous membrane, is infiltrated with cells. There are a number of amœbæ at the edge of the cellular infiltration, and the patch evidently represents an area of extension. In some of these areas of cell infiltration, at a distance from the wall of the ulcer, most of the cells are pus cells; but in others they are almost entirely lymphocytes. In Fig. 7, Plate I, which represents a section through a place where the submucosa is enormously swollen, there are numerous areas of pus cells, with softening of the tissue and abscess formation. Amœbæ are not so often found in these places where suppuration is going on as they are in the areas of infiltration with uninuclear round cells.

There are numbers of bacteria found in all of the purulent foci. In the pus of the ulcer cavity (Plate IV, Fig. 1) there are clumps and masses of micrococci in the pus (*d*), and the granular material going from the cavity through the opening to the surface is almost entirely composed of these. In addition to these micrococci there are short thick bacilli, conforming in their morphology to the colon bacilli; and in one place, in the lowest layer of the mucous membrane, there were found long thin bacilli in the glands and in the interglandular tissue. None of these except the micrococci seemed to have any definite relation to the pathological processes. In many of the ulcers there was a complete absence of suppuration. The contents of these were masses of uninuclear lymphoid cells and large connective tissue cells, and the ulcer was lined with granulation tissue which extended for some distance into the surrounding tissue. One case was entirely free from any complication with bacteria, and in this the relation between the amœbæ and the pathological processes could be clearly studied. In all of the ulcers in this case there were almost no pus cells either in the edges or in the exudation on the surface. Plate IV, Fig. 2 shows a small ulcer from this case. The undermining of the mucous membrane in this case is not so marked as in the preceding cases, the ulcer representing an excavation extending a short distance into the submucous tissue. This tissue is enormously thickened, being as wide as all the other coats combined.

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The cells are large and swollen, the blood vessels distended, and there is considerable round-cell infiltration, most marked at the edge of the ulcer but extending into the surrounding tissue. Immediately in the edge of the ulcer there are a few pus cells, and some of the dilated vessels contain them. The amœbæ are very numerous in this tissue. They are found everywhere in the submucous tissue and extend down to the muscular coat. In Plate III, Figs. 1 and 2 show them under a high power. Fig. 1 is taken from the lower layer of the submucous tissue. They are in a lymphatic at Fig. 1, *a*, *c*, and in the lymph spaces of the tissue (Fig. 2, *a*, *a*). There is a collection of ganglion cells (Fig. 2, *d*) at this point which shows the comparative size of the amœbæ. In Fig. 1 there are two lymphatic vessels which contain a number of the amœbæ. There is a rupture of the wall of one of the vessels, and amœbæ are seen apparently passing from this into the tissues. They are also in the muscularis mucosae, which is swollen at this point; the muscle cells are forced apart; many of them do not stain, and there is an infiltration of round cells between them. Amœbæ are also found in the interglandular tissue of the mucous membrane. In the lymphatics there is a small amount of fibrin and some granular detritus. Wherever the amœbæ are found there is the same swelling of the tissue, infiltration with round cells, and in many cases fibrin formation. As a rule there is no necrosis of tissue in their immediate vicinity; but in a few instances the connective tissue cells around them have no nuclei, and here and there some nuclear fragmentation is found. Sections hardened in Flemming's solution showed an intense fatty degeneration of the cells surrounding the ulcer. This extended to quite a distance from the edge of the ulcer, and all the swollen connective tissue cells in the submucosa contained large amounts of fat. In the tissue the amœbæ usually lay in the lymph spaces, and were surrounded by a clear space, probably from shrinking, due to the hardening agent. They were also found in the lymphatics, and more rarely in the blood vessels, (here in the small veins). Most of the blood vessels in which they were found appeared thrombosed. They contained fibrin and numbers of granulation cells, both in their walls and in the lumen. In many instances they were found within apparently unaltered blood vessels; in some cases blood corpuscles being in the vessel within them.

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The greatest number of amœbæ found in any situation was in the base of a deep ulcer in Case X, Fig. 4, Plate III. This ulcer was a large one, and in one place extended down into the fat of the mesocolon. The amœbæ were found in considerable numbers in the submucous and muscular tissues, where the ulcer had not penetrated through these coats; but the greatest number of them was in the fat. This tissue was necrotic, and there was some hemorrhage into it. Amœbæ in great numbers were in this tissue, both in the immediate base of the ulcer and deeper down in the fat. They were generally not in definite groups, but scattered through the tissue. One thing was striking in connection with the lesions of the tissue and the amœbæ. As a rule they were most numerous in the very smallest ulcers where the process was rapidly advancing, and where there was no suppuration. Either they were found simply scattered through the tissue, or they seemed to form as it were the advance guard of the foci of cellular infiltration. This was particularly well shown in the focus of cellular infiltration in Plate I, Fig. 1, and in Plate I, Fig. 5. They were both in the tissue around the cellular infiltration and just at the edge of this. In sections parallel to the surface they seemed to be in the greatest numbers just beneath the muscularis mucosae.

It was impossible to say just how the ulcers started. It is possible that the small glandular cysts were the starting point of the process; but it seemed more probable that the amœbæ reached the submucous tissue, producing but few lesions in the mucous membrane; and that the essential changes were produced in the submucosa, the mucous membrane being attacked from below. So far as could be seen the changes which they produced in this were an œdema with some fibrin formation and occasionally necrosis. The œdematous tissue then softened, the mucous membrane was broken through and the ulcer formed.

The reaction on the part of the tissue is shown, not in the formation of the pus but in the formation of granulation tissue. When suppuration is a prominent feature in the process, it seems due, not to the action of the amœbæ but to pus-organisms which have entered the tissue. The amœbæ can extend in the tissue a considerable distance, the tissue constantly softening and breaking down behind them. In this case the large ulcers with soft undermined edges will be formed.

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Sometimes there seems to be great undermining before any breaking down of the overlying tissues takes place, and then the large sloughs are formed. In other cases the entire surrounding tissue containing the amœbæ, with the overlying tissue, softens and is broken down, the amœbæ are cast off with the softened tissue, and an ulcer with smooth sides and base results. Or the suppuration of the tissue produced by the bacteria may get the upper hand, and an ordinary suppurating ulcer be the result; the amœbæ being either cast out by the purulent softening, or possibly destroyed by the pus products or the bacteria. In any case there seemed to be an antagonism between suppuration and the amœbæ.

There is a peculiar appearance frequently found in the bases of the ulcers, particularly in those in which active advance seems to have stopped. This was best seen in the ulcers in Case X. The base and sides of the ulcers were lined with a peculiar refractive homogeneous material (Fig. 2, Plate II). This stains deeply with eosin, and has a remarkably bright yellow color when stained with picric acid. It consists of narrow highly-refractive bands arranged in the form of a reticulum. The meshes of this reticulum are oval, have about the same size, and contain both leucocytes and lymphoid cells. Frequently the spaces in the reticulum have not sharp angles, such as would be produced simply by the meeting of the fibres, but are rounded at one end, looking more like a series of loops. These loops seem to extend from various points. This reticulum was figured and described by Kelsch and Kiener,[33] and was regarded by them as a diphtheritic membrane. Although at first it seems to bear a resemblance to canalised fibrin, it certainly is not this, for not only is it too refractive, but it has not any of the staining reactions of fibrin. In sections stained with Weigert's fibrin-stain it remains uncolored. It forms a smooth covering to the edge of the ulcer, and extends in places some little distance into the tissue beneath. It especially involves the walls of vessels in its vicinity. In one instance it was noticed that the wall of quite a large artery near the surface of the ulcer was completely converted into this material. It seems to be a form of degeneration of the connective tissue.

Both when the ulcer extended down to the muscular coat and at a distance from this changes were seen in the muscle. These changes were also best marked in Case X. The muscle is swollen, the con-

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nective tissue septa between the bundles are wider and infiltrated with cells; the muscular fibres lose their nuclei and are converted into a homogeneous hyaline material. In some cases there are comparatively large areas in the muscle which have undergone this change (Fig. 5, Plate II). In others, only single fibres or groups of fibres are affected, and the cross-sections of the muscle have a reticular appearance, the unaltered muscle fibres forming a net work in the hyaline tissue. This does not seem to be due to the direct action of the amœbæ, for it was frequently found below deep clean ulcers where there were no amœbæ. When the base of the ulcer extended to the muscular coat, the reticulum in the base passed directly into the muscle. The changes in the muscle seem different from the formation of the reticulum.

Plasma cells are rather frequently met with in the thickened submucosa. There are other cells frequently found which seem somewhat of the nature of plasma cells, but have a totally different stain from these. The change in these cells consists in the conversion of the protoplasm into hyaline balls, sometimes looking like clusters of grapes. These masses stain in all respects like the hyaline. The small masses vary in size. Sometimes they are very numerous and no larger than the granules in the plasma cells; sometimes the whole cell seems to be converted into two or three round hyaline masses. Many of these were without a nucleus, but frequently the nucleus was found at one side of the hyaline mass. These cells were frequently found in the submucosa, but were more common in the interglandular tissue of the mucous membrane. They are met with in other situations, and in other forms of dysentery, but they seem more common in these cases than in any others.

Changes are also found in the blood vessels of the tissue. These are frequently thrombosed, and the arteries near the larger ulcers show an obliterating endarteritis. In some cases the same œdematous condition of the submucous tissue seems to extend to the walls of the arteries. They are thickened, infiltrated with round cells, and there is an active proliferation of the cells of the endothelium. In some cases there was a new formation of capillary vessels.

In but one case was there any involvement of the small intestine in the ulcerative process. In Case X the ulcers extended a short distance up the ileum. They had the same general character as

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those in the large intestine, but there were few of them in which the process seemed to be active. Microscopically, the surface of the ulcers was covered with the hyaline reticulum, and amœbæ were found in the tissue beneath and around them.

In all of the lesions we have described so far no mention has been made of the solitary follicles. The pathological changes in this form of dysentery approach most closely the form which has been described as follicular colitis. In none of the cases save one was there any involvement of the follicles, and in no case did the process start in these. The follicles were found in some of the sections, and were entirely unchanged, except where they bordered immediately on the ulceration; there they were swollen, and there were accumulations of cells around them. The only part they played in the process was a passive one. In one case, they were distinctly enlarged even at a distance from the ulcer. This was Case VIII, and in this the dysenteric process was complicated by a diphtheritic enteritis. It was only after a prolonged search that amœbæ were found in the submucous tissue about many of the ulcers. This tissue was swollen, but not to the extent found in most of the other cases. The ulcers were covered by a dense granulation tissue, and the follicles were greatly swollen. The cellular infiltration about many of the follicles extended through the muscularis mucosæ and into the lower portion of the mucous membrane. When the muscular tissue was obscured or destroyed by the infiltration with round cells which extended between the glands, appearances were produced of a proliferation of the glands and their extension into the lymph nodule. In none of the sections was there that direct growth of the epithelium into the follicles which was first described by Cornil,[33] and more fully by Woodward.[34] It is possible that the swelling of the glands in this was due to the acute process in the small intestine, which was probably to some extent felt also in the large.

It is probable that this prevented the multiplication of the amœbæ during life in the contents of the intestine and in the ulcers. That there is some multiplication of the amœbæ in the intestinal contents during life seems probable, for they are frequently present in such numbers that they could not be accounted for by supposing that they were set free by the softening of the tissue. Unfortunately, none of the small intestine in this case was preserved for histological study.

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In Case IV, the amœbæ were not found at the autopsy, nor in the further histological examination of the tissues; and they were not found in the stools during life, but only in the sputa and subsequently in the contents of the liver abscess after operation. It is possible in this case that there were very few in the tissues, and they escaped observation.

In the intestine of Case VIII, a peculiar condition was found in the connective tissue cells of the submucosa. The tissues were hardened in Müller's fluid and stained in hæmatoxylin and eosin. Inside of the swollen connective tissue cells were bodies staining with eosin. In some cases these bodies seemed to lie in a cavity inside the cell. They varied from the size of a body a little larger than a white corpuscle to a body almost entirely filling up the cell. In some cases, but a small ring of protoplasm of the cell surrounded the body. They stained brightly with eosin like the amœbæ after hardening in Müller's fluid. They were granular and contained numerous vacuoles. It is not impossible to suppose that these bodies in the cells were amœbæ which had entered the cell when very small, and developed in it. It is also possible that they represented a circumscribed necrosis of the cell, although there is no analogy for such a theory. They were only found in the one case, and as the tissues in this were imperfectly hardened and stained very badly, it was not possible to form any definite conclusions regarding them. In Case IV, many of these connective tissue cells contained large vacuoles, as though resulting from the dropping out of a portion of the cell contents.

9. *Abscesses and other Alterations in the Liver.*

Six of the cases of dysentery in which an autopsy was made were complicated by abscess of the liver. In four of these cases, there was more than one abscess; and in one, the abscesses were so numerous and small, that they resembled more, at first glance, multiple tumor metastases than abscesses. In all of the cases, the right lobe was affected; and in two, there were also abscesses in the left lobe. The most common seat of the abscesses was the right lobe, and there were certain places of predilection in this. The abscesses were usually found near the surface, and either on the lower border of the right lobe corresponding to the hepatic flexure of the colon, or on the upper

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surface close to, or within, the suspensory ligament. In this situation, the abscess generally extended through the diaphragm and into the lung.

The contents of the liver abscesses and the character of the wall varied greatly, both seeming to depend to a great extent on the chronicity of the process. In one case, the entire process of abscess formation could be studied, for there were abscesses in this which were microscopic in size. In the same case there were abscesses of older date, and almost every transition, from the commencing softening to old abscesses with fibrous walls, could be studied in the same liver. The contents of these abscesses vary greatly. In the most recent, the abscess does not empty itself on section. A small amount of glairy semi-transparent fluid exudes and leaves behind an irregular sponge-like mass, the fluid being apparently held in the meshes. In the older abscesses, the contents are more fluid. The fluid has a greyish opaque color, and suspended in it are more solid masses of tissue. In certain cases, the contents were brownish or streaked with brownish-red from admixture of blood. The character of the wall varied. In Case X, in which some of the abscesses were so small, so numerous and apparently of such recent origin, there was no distinct wall to the abscess. In Case XV, the character of the abscesses differed much from the other cases. In this case there were numerous abscesses scattered through the liver and on the surface. They varied in size from 1–5 mm. and on section they had rather the dry caseous appearance of tubercles than of true abscesses.

In some older abscesses, there seemed to be a sharper separation of the liver tissue from the abscess, but still there was little or no formation of connective tissue. Other abscesses were lined with a distinct connective tissue layer, which sometimes was dense and hard, at others, soft and œdematous. Even in the same abscess the character of the wall differs. In some places, it is distinct and fibrous, in others, there are large foci of necrotic tissue extending from the abscess into the surrounding liver. Some of these foci extended a considerable distance into the liver, and formed what could almost be regarded as secondary abscesses. Small abscesses were sometimes in the neighborhood of the large, and were connected with these by a small area of necrotic tissue. The liver tissue in the neighborhood of the large fibrous abscesses was always congested, the lobules indistinct and the tissue

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firmer than normal. In the neighborhood of the small recently-formed abscesses, it was often paler than normal. There was no constant appearance of the liver outside of the area influenced by the abscesses.

The liver was generally of normal size without reckoning the abscesses, or in some cases, larger than normal. In some of the cases it was dark and congested, in others it was very pale. Portions of the liver for microscopic examination were hardened in Müller's fluid, alcohol, chromic acid and Flemming's solution. By each method of hardening, certain distinctive features were brought out which were not so well shown in the others. Some of the pieces were hardened in Müller's fluid which contained a much greater proportion of bichromate of potash than in the formula. They were kept in this in the warm chamber for three days, then washed out and hardened in alcohol. This strong Müller's fluid, used in the warm chamber, proved one of the best means for the preservation of the amœbæ, though it was not so good for the tissues.

The forms of liver abscess could be divided into the small very acute abscess, the larger abscess with a partially fibrous wall, and the chronic abscess with hard dense fibrous walls. The microscopic character of the contents of the abscesses varied. The smaller abscesses contained a tolerably viscid translucent liquid, which contained very few pus or lymphoid cells. There was a good deal of fatty granular material, occasionally a compound granular corpuscle, and small irregular masses which could be recognized as distorted liver cells, or fragments of these. There were very few red blood corpuscles. The amœbæ were numerous and in active motion in all these small abscesses. In the large abscesses the fluid was not so translucent, and its opacity was due not to the presence of pus cells, but to fat, fragments of tissue, and fragmented liver cells. In none of the amœbæ abscesses examined was the fluid of such a character that it could be considered as pus. Here and there, a pus cell was found, but in general, there was a remarkable absence of well-preserved cells. In some of the larger abscesses there were many more red corpuscles than in the small. In Case XV, the abscesses contained a considerable amount of pus cells both in the contents of the abscess and infiltrating the wall.

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The small abscesses in all the sections were hardened with the contents in situ, and embedded in both celloidin and paraffin. Sections were made across the entire abscess, showing in one section the contents of the abscess, the wall, and the surrounding tissue. When these sections are examined with the naked eye or a hand lens, no distinct boundary to the abscess can be seen. The contents are pale, somewhat granular, and within the mass there are more darkly stained, sharply circumscribed portions. Some of the solid masses in the abscess are connected with the wall; others are isolated. They vary considerably in size. Irregular extensions of the abscess into the surrounding liver tissue are often seen, particularly in the very small abscesses. On microscopic examination the general contents of the abscess are a finely granular material, in which cell fragments and small brightly stained particles, resulting from nuclear fragmentation, can be distinguished. There is, however, less nuclear fragmentation than we would expect from such extensive disintegration of tissue. Here and there, a red blood corpuscle and some scattered leucocytes are found. There are some irregular fibres in various places, especially near the edge of the abscess. Some of this is fibrin, as was shown by staining the sections hardened in alcohol with Weigert's fibrin-stain, but most of it represents remains of the connective tissue of the liver.

The solid masses referred to are areas of liver tissue which have resisted the process of destruction. Sometimes one or two of these masses are connected together or with the wall of the abscess, but in general they are completely isolated. They are in great part composed of distinct fibrous tissue with small areas of round cell infiltration. The connective tissue corpuscles and the round granulation cells stain distinctly. The connective tissue is loose in texture, and in some places almost hyaline. It contains an artery, vein, and sometimes one, sometimes several bile ducts. There does not seem to be any circulation in either artery or vein. Sometimes they are collapsed, or they contain a granular mass. The bile ducts are small; they have a low cylindrical or cuboidal epithelium, and are similar to the so-called newly-formed bile ducts in the cirrhotic liver. On the outside of this there are some remains of liver tissue—generally one or two rows of liver cells. The nuclei of some of these cells—the furthest within the mass—are stained; but the outside rows of

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cells are irregular, have no nuclei and gradually merge into the surrounding abscess. These solid masses are evidently small areas of liver tissue around the portal spaces which have resisted the necrosis which has destroyed the intervening tissue. It is impossible to say whether all the portal tissue is preserved in the abscess, or only scattered islands of it. Many of them are certainly isolated, and they are too few to lead us to suppose that they represent the entire portal tissue of the liver which has been broken down in the formation of abscess, unless we suppose that the abscess represents a larger area than the area of liver tissue for which it is substituted. It is remarkable that this tissue should have persisted in the midst of the abscess, when it was certainly cut off from all blood supply and surrounded by the same deleterious influences to which the other tissue had succumbed. These masses are best marked and most numerous in the very smallest abscesses; but even in abscesses 2 cm. in diameter they are frequently found.

There are numerous peculiar bodies which can be recognized as necrotic liver cells, or fragments of these, in the abscess contents, particularly near the sides of the abscess and about the portal remains. The form of these varies greatly. They are generally long and thin, with one or both ends pointed, sometimes of very irregular shape; often they contain vacuoles, or a deep depression in one side like a ruptured vacuole. They have no nucleus; they are finely granular and stain brightly with eosin; and their edges are sharp and refractive, much more so than normal liver cells (Fig. 3, Plate IV, *b*).

The wall of these small abscesses is peculiar. It cannot be said that there is a definite wall. The liver tissue passes gradually into the abscess, and the contour of the edge is most irregular, sometimes extending into the liver tissue for a distance of several lobules. In all cases, wherever the abscess encroaches upon the intralobular tissue, this gives away before the destructive process, and the abscess extends further. The portal tissue offers much more resistance, and the abscess extends around it. In this way the solid masses of tissue in the abscess contents are produced.

Sometimes liver cells with stained nuclei are almost in contact with the abscess contents; sometimes they are separated from it by a considerable area of necrotic tissue. The processes of disintegration in the surrounding tissue can be followed. The capillaries are dilated.

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Sometimes they were filled with blood; but near the abscess the granular contents of the abscess seemed to extend into them. The capillary walls are generally separated from the beam work of liver cells, and sometimes the entire capillary lies apparently in the dilated space which it formerly filled. The liver cells are much thinner. Sometimes groups of several, sometimes a considerable number retaining the form of the tissue, are separated from the wall. They are much more refractive than the normal cell, and contain numerous vacuoles. The specimens hardened in Flemming show that many of these apparent vacuoles are filled with fat; still the process is by no means a fatty degeneration of the tissue. In all the osmic acid specimens there is but little fat shown in the abscess itself. This process of destruction of the liver cells in the periphery of the abscess produced the single necrotic liver cells scattered through the abscess. Frequently the entire abscess and the solid masses in the interior are surrounded by a line of highly refractive reticular tissue. This stains brightly with eosin and picric acid, and gives the same reactions generally as did the tissue in the bases of some of the ulcers. It is not, however, so definitely arranged in a reticular form. Both the liver cells and the connective tissue of the liver capillary walls seem to pass directly into it.

In but one of these cases, Case XV, is there anything which could be regarded as an acute inflammatory reaction in the surrounding liver tissue. There is some slight increase in the connective tissue of the portal spaces when they are near the abscess wall, with slight round cell infiltration. There is no accumulation of leucocytes in this tissue or in the liver capillaries. Occasionally, a few leucocytes are seen in the necrotic tissue, or even in the necrotic cells themselves, but they are very few in number. In the capillaries which are filled with blood, there are no more leucocytes than would be found in the same quantity of blood. In Case XV in which the abscesses were very small and evidently of very acute origin (death having taken place six weeks after the beginning of the attack) there was a very acute inflammatory reaction around some of the abscesses. The capillaries contained numerous leucocytes which also were contained in the necrotic liver cells. The amœbæ were very abundant in these small abscesses.

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Amœbæ are found in the tissue in great numbers; they are scattered generally through the contents of the abscess, but are more abundant in the periphery of the abscess and around the included portal tissue, than elsewhere. In the edge of the abscess they are chiefly found in the capillaries. They do not extend deeply in the surrounding liver tissue, and generally speaking, not beyond the area of necrosis, although occasionally, one or two would be found in the capillaries which are surrounded by normal liver cells. Some of the solid masses in the centre are almost surrounded by amœbæ, but these do not generally penetrate into the connective tissue in the centre of the mass. The amœbæ in the different abscesses vary considerably in number, and are relatively more numerous in the smaller than in the larger abscesses.

Some abscesses are found of microscopic size, which are not connected in any way with the larger abscesses. In these very small abscesses the same process of destruction and extension is seen as in the large. It seems evident that the abscess commences in the intralobular tissue; and one abscess was found in which the softening was confined to the intralobular tissue of a single lobule, extending partly into another. This abscess was near a rather large portal vein, with a slight amount of connective tissue along it. There are several amœbæ in the abscess, and they extend from this into the tissue along the vein, which is swollen and partly hyaline. These small abscesses found in Case X are generally seated immediately beneath the capsule. Sometimes they are separated from this by a narrow margin of hepatic tissue, but more frequently they are immediately beneath the capsule, which at this point is generally slightly thickened.

The alterations of the liver in Case X are not confined to the formation of abscesses. They are rather more marked in the neighborhood of the abscesses, but are met with in the liver generally. The change is a most remarkable one, and consists in extensive necrosis of the liver cells around the central veins. This is most apparent in sections of tissue hardened in alcohol and stained with picrocarmine. When such sections are examined with the naked eye or a hand lens, they show a more or less connected red net-work with yellow tissue between the meshes. Similar sections stained with methylene blue and eosin show a blue net-work with red masses.

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With a higher power the red tissue in the picrocarmine specimens, and the blue in those stained with methylene blue and eosin, are found to be the tissue around the portal vein. The yellow tissue in the one, and the red in the other, is the tissue in the centre of the lobules, which is wholly necrotic. The nuclei of the liver cells do not stain; the cells themselves are highly refractive, and stain an intense bright yellow in picric acid, and a deep red with eosin. Sometimes the shape of the cells is fairly well preserved, and they contain numerous vacuoles. Sometimes they are broken up into smaller and larger segments. The connection of the cells with one another is broken up, and they lie as individual masses between the capillaries. Sometimes the capillaries are perfectly distinct in these masses of necrotic cells, but frequently they are ruptured, and their outlines cannot be made out. The beam work of the liver tissue is dilated, and the broken up cells lie in the spaces. This change is most marked in the sections which were made from the lower part of the right lobe. It is everywhere more or less apparent, but is more marked in the tissue around the central vein of the lobule, and extends a varying distance to the periphery of the lobule, but never completely involves an entire lobule. When neighboring lobules come together it extends from one into the other. The limit of the necrosis is a tolerably sharp one when the specimens are examined with a low power. When examined with a high power, the first change in the cells seems to be a greater refraction of the protoplasm. At the same time the nucleus loses its characteristic epithelial character and becomes homogeneous. Then all trace of the nucleus disappears, and the cell becomes changed into a solid refractive mass. The contrast in the staining between these necrotic cells and the adjoining unaltered cells is most striking; and even in unstained specimens the centres of the lobules stand out prominently. Occasionally a single necrotic cell, or several of them are found in the peripheral tissue surrounded by unaltered cells; but as a rule the necrotic portion is sharply circumscribed. All of the nuclei in the necrotic portion are not destroyed. In one case there were single necrotic cells and small collections of them scattered through the liver without any reference to a particular location in the lobules. The nuclei along the capillaries are unaltered, and their cells seem to be swollen. There are other cells which occupy a peculiar rela-

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tion to the necrotic cells. These are the leucocytes. They are present in the necrotic area in considerable numbers. They are both in the vessels and in the rows of necrotic cells, where they lie between the cell fragments, and occasionally in the necrotic cells. They are generally larger and more granular than normal.

This change in the liver tissue is not due to the presence of amœbæ in the tissue. These are never found except in the abscesses, or in the immediately adjacent liver tissue, where their influence in producing softening and disintegration of the tissue is apparent. This change is also one which could not be attributed to an anæmia of the tissue, produced either from pressure of the abscesses or in some other way.

The small abscesses, unlike the large, do not seem to exert any pressure on the surrounding tissue. Another feature of interest is that the necrotic cells, when the tissue becomes broken down, are taken up by the blood current and carried to a distance. These emboli of necrotic liver cells were frequently found in sections of the hepatic veins which were filled with blood, and around which there was no disintegration of the tissue. They were never found in the vessels of the lung, though it is very probable that they were carried there by the circulation.

These necrotic areas of tissue seem to offer an especially favorable place for the extension of the abscesses. The necrotic cells in the neighborhood of the abscesses rapidly lose their shape, soften, and become a part of the general abscess contents. The softening extends into the tissue and around the unaltered portal tissue, which is in this way separated from the surrounding tissue and forms a solid mass in the abscess. The absence of the amœbæ, and the fact that the necroses do not depend upon their immediate action, cannot be too strongly insisted upon. The action of the amœbæ seems to be shown in the breaking down and actual liquefaction of this already necrotic tissue. The amœbæ are never found except in connection with the softening of the tissue. Even in the microscopic abscesses which are confined to a single lobule, or a very small number of lobules, the amœbæ are always found. Although we now recognize this necrosis of the liver tissue as forming a part, and an important one, in the pathology of various infectious diseases, we know of none in which the lesions are so extensive as they are here. Probably the nearest

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approach to this in the extent of the lesions is found in the liver in cases of yellow fever, which have been described by Councilman.[35] Here the lesions are much more diffuse and not so confined to a definite area in the lobule, although more marked in the intralobular tissue. The process of necrosis in the liver cells is the same in both. In both they are converted into highly refractive hyaline masses, which stain intensely with picric acid and eosin. In the liver, in yellow fever, the contour of the cells is preserved; they do not have the tendency to break up into smaller masses, nor are there any areas of softening in the tissue. Plate IV, Fig. 3 and 4, Plate V.

There is another interesting change in the liver. Certain areas are found in which the liver cells are very small, more numerous, and both the nuclei and the protoplasm of the cells stain more intensely. They are always around the periphery of the lobule, and not in every lobule. In some sections they are much more apparent than in others. These areas are evidently to be considered as foci of regeneration of liver cells.

In sections hardened in Flemming's solution, there are many points in the lesions clearly brought out which were not so apparent with other methods of hardening. In the periphery of the pieces, where the tissue was instantly fixed by the coagulating power of the fluid, it was seen that the separation of the liver cells from the capillary walls extended beyond the necrotic area; and within this space, and among the necrotic liver cells, there were numerous leucocytes, many of which were filled with fat drops.

We believe that the lesions we have described here are the essential steps in the formation of the abscesses of the liver which accompany this form of dysentery. These are not abscesses in the ordinary sense of the word, nor is the fluid contained in them pus. The presence of pus cells is to be considered rather an accidental than an essential feature in the process. There is no suppuration and no inflammatory reaction in the surrounding tissue in this early stage. It is simply a softening and liquefaction of the tissue which is already, for the most part, necrotic. The pus cells which are included in the abscess contents are simply the cells already present in the necrotic tissue. Most of them probably undergo the same liquefaction as the necrotic cells, but some of them are preserved. Although we know that this liquefaction of the tissue plays an

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important rôle in every suppurative process, it never reaches the same extent that it does in this; and this is due to the action of the amœbæ.

Although the more chronic abscesses (Plate VI, Fig. 1) differ from the acute in many respects, just as do the various forms of dysenteric ulceration, they have the same features in common, and the differences can be explained probably by a smaller number and less intense action of the amœbæ, and a minor degree of the diffuse necrosis of the tissue.

From these smaller abscesses various transitions to the large fibrous abscesses were found. In some of these larger abscesses short bacilli, of the same character as those in the ulcers in the colon, were found, but they did not seem to exert any marked influence on the lesions. In general, sections of the walls show on the interior a mass of granular necrotic material which contains in it fragments of fibres. Back of this in many cases, but not in all, there is a sharp line of the refractive reticulum spoken of before. This varies in thickness, and in some places extends deeply into the wall. Small masses of it may also be seen apparently not connected with the surface. There is next an area of granulation tissue composed of uninuclear round cells, which extends into the reticulum and into the necrotic granular mass, the nuclei gradually losing their power of staining. This granulation tissue forms a tolerably well-marked zone, extending in some places deeply into the fibrous tissue beneath. The fibrous tissue contains numerous spindle-shaped cells, and both a diffuse small cell infiltration and circumscribed masses of small cells. In most cases there are very few pus cells, either in the edge of the abscess or deeper down in the tissue. There are numerous bile ducts with low cylindrical or cuboidal epithelium in the connective tissue. They are sometimes so numerous as to form a branching plexus. The blood vessels are numerous and are generally distended with blood.

Liver cells variously altered begin to appear near the margin of the abscess. Frequently they are enormously enlarged, three or four times the size of the normal cells, and are so filled with fat that the protoplasm seems to form a reticulum in the cell. The nucleus stains clearly, and from this the character of the cell may be made out. These cells are at first met with either singly or in groups of two or three, often in spaces in the tissue, and generally the spaces run perpendicular to the abscess wall. Lower down, the liver cells become

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more numerous, and form distinct masses separated by bands of connective tissue. The connective tissue is especially rich in round cells, and extends deeply into the surrounding liver tissue. There is generally some hemorrhage into the connective tissue and in the surrounding liver tissue. The vessels of this are distended with blood, and the rows of liver cells compressed. In some cases, there is evidence of great pressure by the abscess on the surrounding liver tissue. The liver cells are flattened and converted into spindle cells. The capillaries are compressed and obliterated, and the tissue bears more resemblance to the tissue of a spindle cell sarcoma, than it does to liver. These compressed liver cells near the abscess are generally necrotic, and their nuclei do not stain. Further down, most of the nuclei stain as in the normal cells. This compression of the tissue is seen at quite a distance from the edge of the abscess, extending often two or more centimetres into the surrounding tissue and gradually becoming less marked. In it there is always a considerable increase in the connective tissue, with small cell infiltration. The connective tissue is both diffuse and in small isolated patches.

There are certain abscesses which show a combination between those with fibrous walls and the small recently formed abscesses. In these certain parts of the wall are distinctly fibrous. In other parts the wall is formed by necrotic tissue, and necrotic processes extend from this into the surrounding liver tissue. Occasionally, in the neighborhood, there are small necrotic foci with central softening, which are entirely isolated from the large abscess. They become connected with this by gradual increase in size and a continuation of the softening process in both directions. These small foci are in great part composed of round granulation cells, and they melt down in the middle just as did the small abscesses first described. The chronic abscesses increase in size in two ways; both by gradual extension of the abscess with a continual new formation of fibrous tissue, and by the formation of separate small abscesses which soon become a part of the larger. The smaller peripheral foci may meet, and in this way cut off larger or smaller areas of tissue adjoining the larger abscesses. The great irregularity in the walls of the large abscesses is produced in this way. The thickness of the fibrous wall of the abscess varies considerably. In some of the largest abscesses there was little or no fibrous tissue, the wall being composed of the flattened and necrotic

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liver cells. In some cases, there is a good deal of fibrin formed, and there may be a wall of canalised fibrin which in places extends deeply into the liver tissue. The necrotic liver cells seem to pass directly into this fibrin and become an integral part of it. In most cases, there are no pus cell in the contents of the large abscesses, and the small foci in the neighborhood, are composed almost entirely of round granulation cells. In one case, that from St. Joseph's Hospital, there were numerous pus cells both in the exudation on the surface and in the surrounding tissue. In the latter place, they lay between the flattened cells, and were very large and granular.

The amœbæ are relatively much fewer in number in these large abscesses than in the small; while in the sections of the small abscesses they were found at the first glance, and in considerable numbers, it was sometimes quite difficult to find them in the large. Often in large sections embracing a considerable area of the abscess, they were not found at all. They were in the necrotic edge of the abscess, and especially in the small foci in the neighborhood. In the latter place they were generally found just at the edge of the mass of round cells. There were very few in the distinctly fibrous tissue of the wall. Here they were generally found in the blood vessels, sometimes occluding these by their numbers and in spaces in the tissue. In one case, there was a fibrinous mass on the edge of the liver, with considerable increase in the connective tissue just beneath it, and from this an intense round cell infiltration, with the formation of fibrin, extended into the liver for a distance of two or three centimetres; and at the end of this, there was disintegration of the tissue and abscess formation. Amœbæ were found both in the small abscesses and in the round cell infiltration leading to this.

The wall of the abscess in the first case, which had opened into the gall-bladder and duodenum, was very different from the other abscesses. It was lined with a ragged mass of necrotic tissue, and on microscopic examination this was found to be filled with masses of pus cells, and to contain very few amœbæ. There was a considerable formation of fibrous tissue in the wall, which was also infiltrated with pus.

In all these cases, there were marked changes in the liver tissue at a distance from the abscess, but they were not so general or so striking as they were in Case X. These changes consisted in a necrosis of the tissue around the central vein, and in a new formation of connective

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tissue. This was often accompanied both by a new formation of bile ducts, and frequently by an enormous dilatation of the existing ones. This was so marked in some places that the liver presented the appearance of cirrhosis. Case XV was distinguished from the other cases by the very irregular distribution of the necrotic tissue. Groups of necrotic liver cells or single ones were irregularly distributed in the tissue without any definite position in the lobule.

Bacteria were found in the abscesses in three, out of the six cases. In one of these, Case XIV, the autopsy was made thirty-six hours after death, and in another, Case II, the tissues lay twelve hours in a mass before being examined and hardened. Bacteria were found in both of these, and in Case III where there was perforation into the duodenum. Cultures were made from the abscesses in two of the cases. In one of them colon bacilli developed, and the other was sterile. Bacilli were found in the sections of the one. In one case, in the necrotic tissue of the wall, there were numbers of different organisms. Some of them conformed to the appearance of the colon bacilli. These lay in the blood vessels in the edge of the abscess and deeper down in the tissue. In most places they seemed to produce no lesions in the tissue around them. They were most numerous in the first case in the liver tissue, to which the colon was adherent. In this there was the most extensive necrosis of the liver cells, and in places a great increase in the connective tissue. The bacilli were chiefly in, or scattered around the vessels of the necrotic area. It could not be said even here, that they produced the necrosis, because they were also found in other parts without any necrosis around them.

When we sum up these changes in the liver, it seems evident that there are two distinct series of lesions. First; the extensive necrosis of the tissue which cannot be regarded as due to the direct action of the amœbæ. In the first place no amœbæ were found in these necrotic areas; and in the second, the lesions were too regular in situation and extent to be due to the action of organisms. We should regard these lesions as due to the action of the soluble chemical products of the amœbæ which were absorbed from the intestinal ulcers. Second; the abscess formation which is due to the direct action of the amœbæ. There were no abscesses in which the amœbæ were not found. They were most numerous in the smallest and most recent of these, and in those situations in which the abscess was extending.

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There were no other organisms in these small abscesses; and even in the large the bacteria which were found did not seem to have any causal connection with the abscess. None of the organisms which we are accustomed to consider pus-organisms, were found. The lesions were of a different character from those produced by bacteria.

10. *Abscess of the Lung.*

Abscess of the lung, secondary to abscess on the upper surface of the liver, was found in three cases. The lung abscesses are not less interesting than those in the liver, and resembled these in several particulars. They were all very similar to each other in their general appearance. All were lined with opaque, ragged, necrotic material, which in places projected in large masses into the cavity. There were also projections from the abscess into the surrounding pulmonary tissue and by the union of these large masses of tissue were cut off as in the large liver abscesses. In a few places, the abscess wall was comparatively smooth, and formed of dense connective tissue. In other places there was no definite boundary between the necrotic tissue lining the abscess and the surrounding lung tissue. In Case X, these differences in the character of the wall, were well shown. The lung adjoining the abscess was very dense in every case. It was solid on section, and of a whitish opaque color, particularly around the abscess; further away it was more greyish and transparent. The cut surface was perfectly smooth, and a large amount of fluid escaped on section. A purulent fluid could be squeezed from some of the bronchi, but most of the fluid which escaped had an œdematous character.

Microscopical examination of the abscess contents, or rather of the contents clinging to the wall—for the abscess was generally almost or entirely empty—shows a great deal of granular detritus, numerous round lymphoid cells, a few pus cells, numbers of red corpuscles, fat drops and amœbæ. Fragments of the lung tissue, in the shape of elastic fibres, were also found. The consolidation embraced the entire lower lobe in which the abscess was found. Portions of the abscess wall were hardened in alcohol—Müller's fluid and Flemming's solution. Sections which embraced the abscess wall and a part of the contents, show on the interior towards the abscess, and forming the contents of this, a granular mass. This is mostly composed of small

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granules which do not stain, and among them larger, brightly staining granules, evidently derived from fragmentation of the nuclei. Most of these are distinctly round, but others are in the form of brightly-staining rods. These are most apparent in sections which had been hardened in chromic acid or Flemming's solution, and stained with safranin. This evidence of nuclear fragmentation was more generally found in the lung than in the liver abscesses.

There are numerous cells distributed through the granular mass of the abscess; they are generally single, but sometimes small groups of them are found. As we approach the edge of the abscess small, isolated masses of tissue are found. For the most part this is nothing but small bits of elastic tissue, though here and there small portions of the lung consisting of one or several air cells are seen. Most of this tissue is completely necrotic, but there are some masses in which the nuclei are stained (Fig. 4, Plate VI). Small masses of fibrin are also seen. The wall of the abscess differs in various places according to the greater or less formation of fibrous tissue. In parts of the wall there is a dense mass of connective tissue, from 2 to 3 mm. thick. This is generally infiltrated with round cells in rows or masses. On the edge there is sometimes seen an approach to the formation of the hyaline reticulum spoken of in connection with the ulcers in the colon and the liver abscesses. In other places there is a dense mass of fibrin in the edge, which in places extends into the fibrous tissue. In other places, which apparently denote a rapid advance of the process, there is no definite wall to the abscess. It extends directly into the adjacent lung tissue, and its boundary is indefinite. Here there were numerous masses of tissue along the wall, partially or completely cut off from the lung (Fig. 4, Plate VI). The granular material extended down into the alveoli. In the tissue, immediately adjoining this part, there is no inflammatory reaction. There are no leucocytes in the tissue or in the blood vessels, and the granulation cells are not increased to any considerable extent.

The lung tissue adjoining the abscess (Fig 2, Plate VI) was in the same condition in all parts, whether separated from the abscess by a zone of fibrous tissue or whether the abscess extended directly into it. The alveoli are very small, and the interstitial tissue of the lung enormously increased. The small size of the alveoli seems to be due entirely to this increase in the connective tissue, for there are no evi-

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dences of compression. The alveoli are very irregular in shape, and frequently appear as very small irregular spaces in the tissue. They are all lined with a distinctly cuboidal epithelium, the cells being in most cases clearly separated from each other. The nuclei are large, round, vesicular, and stained brightly, so that in places the tissue presents rather the appearance of an alveolar glandular tissue than the lung. In the sections which were hardened with Flemming's solution and stained in safranin, there are numerous nuclear figures in the epithelial cells. The contents of these alveoli vary greatly. Immediately around the abscess they are filled with large round cells, many of them fully as large as the amœbæ, which they greatly resemble. These cells when examined in the fresh state are filled with refractive fat granules. In the specimens hardened in Flemming, these granules give the reaction of fat. The nucleus stains brightly and is often irregular in shape like the nucleus of a leucocyte, but in other cells it is distinctly round and of a more epithelial character. In some of the cells there are round, smooth, hyaline-looking masses. Among these large cells there are numerous granulation cells and a few scattered leucocytes. Small masses of fibrin are occasionally found here, but the fibrin is more abundant in the alveoli, a short distance from the abscess wall.

Many of these alveoli are filled with a dense mass of fibrin, composed of thick, irregular-shaped filaments. In some cases, it is in the form of large, irregular masses, not at all filamentous in character. It gives all of the staining reactions for fibrin. The interstitial tissue, as said, is greatly increased, and this forms a part of the contents of the alveoli (Fig. 2, Plate VI). The walls of the alveoli are greatly thickened, and the connective tissue projects in the form of bud-like masses into the alveoli, in some cases completely filling them up. The fibres of this tissue are loose and separated from each other; the cells are comparatively few in number. Granulation cells are found in it in varying numbers, in some cases being abundant and grouped in large masses; in other places only a few scattered ones are found. The projections into the alveoli in many cases, pushed the epithelium before them, and the projections were covered with the cuboidal epithelium like the other parts of the wall. In some cases, in the middle of an alveolus, a round mass of connective tissue covered with epithelium is found without any apparent connection

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with the wall. This evidently results from the section of one of these projections which had entered the alveolus either above, or below the plane of section. In other cases, the tissue grows into the alveoli from the wall, and is not covered with epithelium, the other contents, generally fibrin, gradually giving way to it. Small blood vessels were frequently found in these masses of connective tissue within the alveoli. There is not only this general connective tissue formation with preservation of the general character of the tissue, but, large masses of connective tissue more or less isolated, are frequently found. These are principally about the larger blood vessels. All this connective tissue has a loose texture, and contains few connective tissue corpuscles. The lobules of the lung are separated from each other by thick bands of this tissue.

The bronchi are much altered; the walls are thickened, and contain great numbers of round cells. Some of the bronchi are filled with fibrin and pus cells; in others, the cells are more lymphoid in character. In nearly all of them, a similar condition to that of the alveoli is found in the projection of masses of connective tissue from the wall (Fig. 2, *b*, Plate VI). In some cases, these are small and bud-like, and are covered with epithelium. In others, the epithelium is absent over some part of the wall, and here there is an ingrowth of connective tissue with blood vessels, which gradually substituted itself for the fibrinous contents. There was one peculiar appearance which often presented itself: that is the presence in the alveoli of dense, concentric, round or oval masses of various size, which seem to have no connection with the alveolar walls. In other cases, in some of the projecting buds of connective tissue, there are no nuclei; the tissue is very dense, and similar to the isolated masses. We regard these as masses of connective tissue which are severed from their connection with the wall, and become altered into these concentric bodies in the same way that the rice bodies are formed in the joints.

The amœbæ varied greatly in numbers, both in the different cases, and in different parts of the same abscess. They were found in the contents of the abscess, and were especially numerous in the places where the abscess is rapidly advancing, and where there is no distinct wall. Fig. 4, Plate VI, shows such a place. The amœbæ are in great numbers in the alveoli immediately adjoining the abscess. They are found in the tissue all around the abscess, but in smaller numbers

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as the distance from the abscess increased. They are not found in the bronchi at a distance from the abscess; but in the bronchi near the abscess, (Fig. 3, Plate VI), they are frequently found in considerable numbers. Here, they are not only in the wall, but in the epithelium and in the lumen. In the fibrous wall of the abscess, they were found in considerable numbers in some sections; in others, they were entirely absent. In this place, like the corresponding part of the liver abscess, they were either in the blood vessels or in the spaces of the tissue.

In the production of both the lung and liver abscesses, the action of the amœbæ appears to be the same. They act by producing a necrosis and liquefaction of the tissue, and in both, the peculiarity of their action and the difference which most clearly separates it from the action of bacteria, especially the abscess-producing ones, is shown in the general absence of acute inflammatory reaction on the part of the surrounding tissue. This is especially the case where they are present in the greatest numbers, and their action is most intense. In other cases in both the liver and lung abscesses, the reaction of the tissue is seen in the production of a fibrous capsule. In all cases, in the intestine, in the liver and in the lung, unless there is a complication in the presence of the pus-producing bacteria, there is an absence of suppuration. The extension of the lung abscess into the surrounding tissue, and the formation of separate foci is probably due to the entry of the amœbæ into the tissue by means of bronchi bordering on the abscess; and to the easy means thus given them for their entry into the tissue, the irregular extension of the abscess and the ragged character of the walls are due.

In three of the cases, the first and the two last, there was peritonitis. In the first case, it was localised in the region of the liver. In the other two cases, there was a general peritonitis. In the first case, no amœbæ were found in the exudation. In the others, they were very numerous. The character of the peritonitis in these cases, at once would strike one as being very different from any of the ordinary forms. It was distinctly fibrinous in character, but instead of the opaque color of this, it had a pale, transparent appearance. This was partly due to the amount of fluid which it contained. It was most abundant over the upper surface of the liver, and the fissure on the edge was filled with it. It could be easily removed. Examined fresh, it contained fibrin, comparatively few leucocytes, large numbers of lymphoid cells

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and large cells with oval epithelial-like nuclei, evidently arising from proliferation of the endothelium. Portions of it were hardened in the various media, and sections made after embedding in celloidin. In these sections, the fibrin was found in comparatively coarse filaments. The cells were principally of the character of lymphoid or granulation cells; but in places, numbers of pus cells were also found. The amœbæ were present in considerable numbers, and bore no definite relation to the other constituents of the exudation.

11. *Summary of the Pathological Anatomy.*

When we now sum up briefly what we have described in detail, we find that this is a disease which is characterized by definite anatomical lesions having a certain uniform character but which differ in some respects in the various organs; the differences being capable of explanation by the differences in anatomical structure of the various organs, the greater number of amœbæ, and the varying intensity of their action, the complication of the action of the amœbæ by the action of other organisms, and the varying degrees of resistance of the tissue. No analogy can be found between the tissue changes produced directly by the amœbæ, and those produced by the bacteria. If in general, we assume the action of the bacteria to be an injury to the tissue, shown either by a slow necrosis with the production of granulation tissue, or a more rapid necrosis with more acute inflammatory changes, and the production of an exudation with fibrin and pus, the action of the amœbæ is different from either. In the intestine, we do not see this action in its purest form, for it is probable that here the process is always more or less complicated by the action of the ever-present bacteria or their products. We have seen it in both the liver and lung abscesses; and here, in its most typical form, and especially in the small abscesses of the liver, there is an absence of any reaction on the part of the surrounding tissue. In the smallest liver abscesses, in Case X, there is never any production of granulation tissue in the immediate vicinity, nor is there any suppuration. There is probably a considerable serous, or in some cases, a sero-fibrinous exudation from the vessels which forms the fluid of the abscess. We would rather regard as the action of the amœbæ both a production of necrosis of the tissue and a solvent action on the intercellular sub-

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stance, and the connective tissue, as well as on the cells of the tissue. There is not the remarkable destruction of cells with the breaking up of nuclei and the formation of nuclear detritus observed, as in the action of certain bacteria. Nuclear detritus is usually present in a certain degree, especially in the abscesses of the lung, but not necessarily so. The necrotic cells seem to undergo a slow destruction and liquefaction, the nucleus sharing the same fate as the cell protoplasm. In all cases, the number of amœbæ found in the tissue bears a distinct relation to the activity of the process. Aside from this direct action of the amœbæ on the tissue, and associated with their presence, we must recognize as probably of equal importance with this, the diffuse necroses of the liver cells, probably the result of the absorption of the chemical products of the amœbae from the intestinal canal. This action would, of course, be felt in its greatest intensity in the liver, and seemed to be almost limited to this. There was no evidence of this action in the lungs, and only slight evidence in the kidneys.

The kidneys were examined carefully in three cases; in two of these after hardening in Flemming's solution. In all three cases, the changes were limited to a cloudy swelling of the tubules, with very slight, fatty degeneration. The glomeruli were unaltered; but in one of the cases, in a few glomeruli crescents of coagulated albumen were found around the vessels.

In the liver, this action of the chemical products of the amœbæ, whether toxic albumens or ptomaines we are unable to say, is seen in the diffuse necrosis of the liver cells, affecting almost equally all parts of the liver. There was some evidence of this in all the cases, but, like all the other lesions, it was best marked in Case X. The necrosis of the tissue in this case was confined to the cells in the center of the lobules—in some cases, confined to only a few cells around the central vein; in others, extending over almost the entire lobule. There was little or no fragmentation of the nuclei; the cells were changed into highly refractive masses; in some cases, preserving their form, in others, broken up into fragments and separated from one another, and lying in confused masses between the capillaries. It is very curious, considering that these products must have entered the liver by the portal vein, and must have first exerted their action on the cells in the periphery of the lobules, that the changes should have

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been limited to the centre of the lobule. A possible explanation might be found in the greater resistance of the peripheral cells, this being produced by a more constant exposure to the action of similar products derived from the intestine. There is more similarity between these lesions of the liver and those found in yellow fever, than in any other diseases. In the latter, however, although the necrosis of tissue with exudation of leucocytes, is the principal or only lesion found in the liver, the necrosis affects only single cells or groups of several; and the necrotic cells are generally almost confined to, or certainly most numerous in the periphery of the lobule. We think it is difficult to over-estimate the importance of these diffuse lesions in the liver. We have never found that they of themselves progressed to complete liquefaction of the tissue and the formation of the so-called abscesses. Wherever the smallest abscesses were found, even when confined to a single lobule, the amœbæ were also present. It is possible to conceive, however, that when the action of the chemical product is very intense, or possibly more intensely felt in certain places, that entire lobules might be affected and softening take place. In this way, abscesses might be formed without the amœbæ, or what seems more probable, these necrotic areas would offer the most suitable soil for the action of any pus organisms which might accidently enter the tissue. Even without the formation of abscesses, this necrosis must produce extensive and important alterations in the tissue. Although the loss of tissue may be wholly, or in great part repaired by the proliferation of liver cells in other places, connective tissue will in great part be substituted for it, and a condition of cirrhosis be produced. It is probable that many of the lesions of the liver which have been so long recognized, especially in India, as an accompaniment or sequence of dysentery, are due to this change in the liver.

In the lungs, this softening of the tissue is also seen, but the lung tissue appears to be more resistant than the soft parenchymatous liver, and there is not the same opportunity for the wandering of the amœbæ into the tissue. Nearly everywhere in the abscesses, there is the same notable absence of suppuration that is seen in all the other lesions. It seems probable that the irregular extension of the abscess, is due to the wandering of the amœbæ into the adjacent lung tissue along the bronchi, which are near, or open into the abscess. The peculiar pneu-

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monia surrounding the abscess, offering as it did the most exquisite example of interstitial pneumonia, we would rather regard as the reaction of the tissue to the abscess, than to any direct action of the amœbæ or their products, on the tissue. Though more extensive, it was very similar to the interstitial processes found in the neighborhood of old phthisical cavities, and making a part of the changes in fibroid phthisis. Accompanying this interstitial process in the lungs and in many cases apparently preceding it, was a more acute inflammation with fibrinous exudation. Though the effect of the absorption of toxic substances from the alimentary canal was so marked in the liver, nothing of this was seen in the lung.

The amœbæ, probably reach the large intestine in food or drink. They exert no action on the stomach or small intestine, for they probably do not find there the suitable conditions for their increase. It is only when they reach the large intestine that they find the conditions favorable; and one of the essential conditions seems to be from the researches of Cunningham, and also in part from our own, that the reaction of the material in which they grow, shall be alkaline. The small intestine seems only to be affected in those cases where there is an enormous number of amœbæ in the large, and they probably enter the small from the large, but they only affect a small section of the ileum.

We must believe that the amœbæ multiply rapidly in the large intestine, and to a much greater degree here, than in the tissues. Apart from the production of definite ulceration, their presence in the large intestine appears to be deleterious, for we have on the surface of the mucous membrane evidence of their action in a superficial necrosis, and softening of the epithelium. There is also an increased secretion of mucus from the epithelial cells. All of the cells show this, and the glands are often filled with mucus. It is probable that the cysts in the mucous membrane result from the dilatation of glands whose mouths have been plugged up by the accumulation of mucus. The ulceration appears generally to be caused by the invasion of the mucous membrane by the amœbæ. They enter this, passing along the interglandular tissue, and in some cases, they enter the glands. They were found in these places both near the ulcers, and at a distance. They pass through the muscular coat, enter the submucosa, and in this, their action becomes most apparent. It is shown here just as

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in the liver abscesses, by a softening of the tissue. There is some hyperæmia, but not nearly as much as we are accustomed to see in croupous dysentery. The submucous tissue becomes œdematous, swells up and softens, and with this softening the ulcerative processes begin. The tissue above, deprived of its blood supply, becomes necrotic and falls in upon the cavity in the submucosa. The follicles are not especially attacked; they simply share the fate of the surrounding tissue. There is some fibrinous exudation into the tissue, and often necrosis of the cells immediately around the amœbæ, but the production of necrosis does not appear to be the primary action of the amœbæ. It is especially in the loose connective tissue, between the coats of the intestine, that the action of the amœbæ is most pronounced. The muscular coat offers some resistance; it is not generally destroyed, but the amœbæ pass through it in certain places, enter the intermuscular tissue, and there play the same part that they did in the submucous tissue, the tissue overlying the infiltration undergoing necrosis in mass. Along with this, there is a peculiar hyaline degeneration of the muscular coat. Not only do the ulcers increase by this continual undermining process, but there is probably a continuous softening and liquefaction of the tissue from the contents of the intestine. The whole typical course and appearance of the ulcer may be completely changed from action of the bacteria in the intestinal canal. Extensive suppuration may take place, and this process may be entirely substituted for the action of the amœbæ.

How do the metastases into the liver take place? The amœbæ are found both in the lymphatics and in the blood vessels; and it would seem on first glance that they are carried into the liver by these channels. They are found so commonly in the lymph spaces and lymphatics of the tissue, that nothing would seem more likely than that they should travel along these canals. This is not the case; for the lymphatic glands, though swollen, are not affected to the same degree that we find in other forms of dysentery. The closest examination of these glands fails to show any amœbæ. The follicles of the glands often show necrosis of the cells with nuclear fragmentation. This is probably due to the action of the soluble products of the amœbæ. If the amœbæ are not found in the lymph glands belonging to the infected tissue, and there are no marked changes in them, the extension of the process along the lymphatics can be excluded.

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The amœbæ are frequently found both in the walls and in the interior of blood vessels; both in those in which the circulation seems to be suspended, and those in which, as far as we can see, the circulation is active. The small veins are the vessels most frequently invaded. There is one point about these abscesses of the liver, which was very striking in our cases, and to which attention has been called by all writers on liver abscess. It is especially the right lobe of the liver which is affected, and there are certain preferred situations in this. These situations are on the under surface of the right lobe, in the neighborhood of the hepatic flexure of the colon, and on the upper surface close beneath the diaphragm. When the left lobe is affected, as was the case in one of our cases, there are generally multiple abscesses scattered all over the liver. If the amœbæ were carried into the liver as emboli from the blood current, it is difficult to see why they should be lodged in any particular part of the liver. The direction of the portal vein would rather favor their lodgment in the right lobe, but it would not explain why the abscesses are so generally superficial. They are also found with equal frequency in the veins of the liver and in the lung, but there is no evidence that they are carried from these organs by the blood current. The abscess of the lung is usually found in one place in the lower lobe, in contact with the diaphragm. If the amœbæ were carried into the lung from the liver by the blood current, there is absolutely no reason to suppose that this part of the lung would be the region affected. Metastases in distant organs do not occur; and they would if the amœbæ were carried along the blood current.

The abscess in the lung is frequently a direct extension of the abscess from the liver into the lung through the diaphragm. In other cases, the diaphragm is left intact. Although the number of cases which we have studied is far too small to make any general statements on this head, it seems to us far more likely that the amœbæ reach the liver by another route than through the blood or lymphatic vessels. This is by means of the abdominal cavity. The amœbæ pass into this from the intestine; they either enter the liver directly from the hepatic flexure of the colon, or they wander or are carried, as other insoluble matters are, along the upper surface of the liver beneath the diaphragm. In the peritoneal cavity they may produce peritonitis, as in Case X; or they may enter the liver appar-

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ently without producing any effect on the peritoneum. One of our cases spoke very clearly in favor of the infection of the liver from the peritoneum. In this there was a circumscribed, fibrinous exudation on the surface of the liver, and some distance beneath this, in the tissue, there was a beginning abscess formation. Although we regard this as probably the most frequent route by which the amœbæ enter the liver, it cannot be the only one. Case XV, which was autopsied after most of this paper was written, shows conclusively that there must be infection through the vessels as well. In no other way could we explain the formation of great numbers of small abscesses both on the surface and in the tissue. These small abscesses were as numerous in the left as in the right lobe of the liver.

Whether or not the amœbæ are capable of producing the abscesses of the liver so often met with in the tropics, without any ulceration of the intestine, must still remain sub judice. Not only must there be the most careful search for intestinal lesions, but it must be definitely proven whether or not amœbæ are found in these abscesses. It would not seem impossible for the amœbæ to enter the liver from the duodenum along the biliary passages. The whole question of these abscesses of the liver is one which should be cleared up; and with the abundant opportunities for their study which are offered in India and other tropical countries, we have no doubt that it will be.

That the amœbæ are the direct cause of these lesions, there appears little doubt. Not only have we the positive evidence of Kartulis of the cultivation of the amœbæ and the production of dysentery by the inoculation of animals with pure cultures, but there seems to us sufficient evidence of their causal connection from the anatomical study of the tissues. We have in this disease lesions which are characteristic, and which differ in their general characters and mode of production from those produced by any organism with which we are acquainted. We constantly find amœbæ associated with these lesions sometimes alone, sometimes associated with bacteria, whose presence we believe to be accidental, and which we know are not of themselves capable of producing similar lesions.

We have thought it would be well, believing that the lesions in this form of dysentery are sufficiently characteristic to enable us to recognize the disease from the description of the ulcers of the colon given by various authors, to go over the more prominent publications

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on dysentery. We have in this devoted principal attention to the French authors in Algiers and the English in India, together with the principal publications on the subject in other countries.

The English literature relating to this subject is mainly derived from the descriptions of army surgeons in India, and elsewhere in the tropics. Among the earlier publications, is the oft cited description of Sir John Pringle.[36] He made four autopsies on soldiers who died of dysentery, or immediately after it. In the first case, there was extensive thickening and ulceration of the large intestine. "The villous coat was either wholly abraded or changed into a corrupted, slimy substance of a greenish color." In Case IV, with extensive destruction of the mucous membrane, there were also several abscesses of the liver. Munro[37] gives the results of autopsies made in dysentery; and says he found the rectum inflamed, partly livid, and black and eroded, especially the internal coat. An interesting autopsy made by Hewson is quoted by Sir John Pringle. "The dissector having cleared away the blood and mucus from the inside of the cæcum, the colon and rectum, he made us take notice of certain excrescences of a lighter color than the rest of the surface. They were of a roundish figure, nearly equal in their height, which was about the one-twelfth of an inch, but of unequal breadth. They were only found in the large intestine." Wollaston[38] describes two cases in which the lesions were very similar to those described by Pringle and Hewson. John Hunter[39] gives an account of the lesions he has observed in autopsies made on dysenteric subjects in Jamaica. In some of the cases, there were small tubercles like pustules scattered over the mucous membrane. Their progress, which he studied by comparing different stages in the same intestine, or in different cases, he considers to be as follows: "There is first formed a small round tubercle of a reddish color, and not larger than one-tenth of an inch in diameter. It gradually increases in size until it reaches one-quarter of an inch in diameter, and grows paler as it enlarges. In this stage there appears a small crack on the top, with a small depression which gradually enlarges. On examining the contents of the little tumor, I have found it a cheese-like substance. The pustule is seated under the villous coat, between that and the muscular coat. As the opening enlarges, the edges become prominent, and the base rough and scabious. From the

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base, matter oozes which is sometimes tinged with blood. Such is the progress of one; but they are often in clusters, and become confluent, so as to form a rough, unequal, ulcerated surface, with a hard and thickened base. Sometimes they appear like a small eating ulcer, in which the prominence of the edges gives an appearance of a loss of substance, as if the villous coat were entirely removed." This description by Hunter might be very well referable to the small elevations described in our cases.

Colin Chisholm [40] describes the intestinal lesions he observed in the West Indies in very much the same way. "The large intestines are always in a state of inflammation. The effects of this are gangrenous blotches, but oftener tuberculous excrescences on the internal surface of the large intestine. These excrescences are the result of an exudation of lymph. The rectum is more especially the seat of them. The whole length of the intestine is often covered with a dark, bloody slime, and a number of these excrescences which resemble very much the pustules of smallpox." Davis [41] (quoted from Woodward), reports forty-two autopsies made on the Walcheren diseases, among them a number made on dysentery. He found in the dysentery following the fever, the coats of the large intestine preternaturally thick and hard, and in places small, round bodies with an ulcer at their point, or little ragged ulcers excavated in the middle, resembling chancres; or one large or a succession of small ulcers spreading wide and deep into the coats of the intestine. He speaks of these eminences as tubercles, and says that they had their origin underneath the villous coat of the intestine. He says that a similar condition was found in the small intestine, so that it seems probable that some of his cases were typhoid.

Annesley,[42] in his description of dysentery in India, makes the division into the uncomplicated and the hepatic dysentery, or dysentery complicated with disease of the liver. The dysentery in these cases he does not believe to be the cause of the liver trouble, but to be the consequence of this, and due to the action upon the intestine of the abnormal biliary secretion. The more acute forms are accompanied by acute affections of the liver, and the more chronic with the formation of abscesses and other organic changes in the substance of the viscus. The connection between the bowel and the liver affection is of two kinds. In one it appears to be altogether a

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symptom of a morbid secretion of bile or of the advanced stage of structural disease of the liver. In the second variety, the hepatic disease seems to be induced by the disorders of the bowels, and more particularly when the disorder is of a subacute or chronic kind. Although the liver and the intestinal disease often supervene one on the other, still they are often simultaneous in their attack, and the priority of one or the other lesions can scarcely be detected. In the acute variety, the dysentery may be said to be due to the action of the disordered bile. Sometimes the intestinal trouble is so pronounced that the preceding hepatic trouble is fully masked by it. Annesley gives a large number of cases of this so-called hepatic dysentery; and from his descriptions of the lesions, they were almost certainly all the amœbæ form. The colon generally does not show any lesions externally. Upon grasping it, and running the fingers along it, a different feeling is communicated to the touch in the different parts. At one place it is thickened and doughy; at another, thin and membranous. The affection often extends to the peritoneum, and a small amount of exudation is seen on the surface of this, or there are adhesions binding the various surfaces together. He describes in several places the abnormalities which have resulted from these adhesions. There are great variations in the calibre of the gut, which is sometimes distended; at others, firmly contracted. The ulcers are sometimes small and thickly clustered, or confluent in one place, and large and single in others. The ulcers may be in the centre of an elevated and thickened base. In some forms, in addition to the ulcers, the coats of the intestine are thickened from congestion and effusion of serum. He explains the reason that the small intestines are not affected by the disordered bile. Owing to their irritability, fluids pass along them more rapidly, and are not so long in contact with their walls. From his description of the lesions of the small intestine in diarrhœa, there is little doubt that he has included among these many cases of typhoid fever. He gives an excellent description of the abscesses of the liver. From the plates and the descriptions, it is evident that he considers, along with the true dysenteric abscesses, those following the portal vein. He describes very accurately the softening of the liver around the abscess, and the ragged character of the walls of some of the abscesses, while in other cases, there is a firm

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fibrinous wall. This work of Annesley is by far the best of the descriptions we have of the Indian dysentery.

Macpherson [43] gives, in tabulated form, a number of autopsies made on the Bengal dysentery. His descriptions of the lesions are very short and unsatisfactory. He does not regard the dysentery of Bengal as different from that of Europe, and finds the lesions similar to those described by Baly and Rokitansky.

Lyons [44] gives an admirable account of the dysentery which he saw in the Crimean war. He describes both the acute and chronic forms. In the acute, he describes the exudation on the surface with but slight thickening of the mucous membrane, and little or no ulceration. It is evident that this acute form he describes was diphtheritic colitis. The chronic forms were characterized by great thickening of the intestine, which was often one-quarter of an inch thick. One form of ulceration he considers to begin in the follicles. "This class of cases, from its morbid anatomy, deserves to be considered as a special variety of the disease; and from certain characters which it presents in a marked manner, we venture to assign to it the term of destructive or pustular, or from its chief anatomical seat, follicular colitis." In this form the colon shows here and there small, prominent, rounded masses, generally about the size of a pea. These little bodies break readily on pressure, and give exit to a small amount of pus. In many cases they contain cavities with jagged, irregular, undermined borders, which are undergoing destruction. These pustules he regards as of the same nature as the tubercles described by Hewson and Pringle.

Parkes [45] divides the lesions which he has observed into three classes. In the first, ulceration occurs in the follicles; in the second, it is around these; and in the third, it is due to effusion of fluid in the submucosa. In most cases, his description agrees with the amœbæ form.

Morehead,[46] in his excellent treatise, gives a large number of autopsies on cases of dysentery and abscess of the liver. Nearly all of the acute cases presented the lesions of diphtheritic colitis. In the chronic cases, there was thickening of the intestine, and the formation of small, undermining abscesses, which seemed to begin as pustules. He speaks of this as submucous suppuration. It is certain that most, if not all, of his chronic cases were the amœbæ form. He also gives many interesting cases of liver abscess. In one case, where the patient

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had dysentery for a long time, there was an abscess formed between the liver and the diaphragm, which penetrated into the lung. In another case, there was abscess of the liver, and an abscess between the lung and diaphragm (sacculated empyema). There was another abscess between the second and third lobes of the same lung, and in the third lobe, an abscess containing about six ounces of pus. The lung tissue around all these abscesses was indurated and hepatized.

Horton,[47] in his description of the lesions found in dysentery, says that the actual point of mischief at the onset of the disease is the areolar tissue below the sub-basement membrane, where exudation takes place, which leads to ulceration, hemorrhage and purulent discharge. He divides the ulcers into transverse and circular. The circular ulcers were found in nearly all cases of dysentery produced by hepatic abscess, he agreeing with Annesley in the connection between dysentery and abscess of the liver.

Maclean [48] agrees with Parkes in considering the follicles as most frequently the starting point of the ulcerations. The suppuration generally begins in and around these. In the beginning they are much enlarged and distended by a whitish material resembling flour and water. As the disease advances, these glands become prominent; they are surrounded by a vascular ring, and ulceration begins either in the apex or around the glands. The mucuous membrane, pressed upon by the hypertrophied gland, has its vitality destroyed and ulcerates, or the ulceration may begin by effusion of fluid beneath the mucous membrane. This effused matter is of a whitish or yellowish color; the mucous membrane is elevated, softened, and easily rubbed off, exposing an ulcer. In acute cases, the cæcum and colon are thickened by exudation between the tissues, and the solitary glands with their white contents and black points, are prominent. The intervening tissue is crowded with ulcers in various stages, and of all sizes. The softened mucous membrane is of a dark color, and presents a more or less mammillated appearance. Maclean thinks that the cases observed by Dr. Shanks and himself, in Secunderabad, were of a much severer type than those seen by Dr. Parkes. There were large ulcers of varying size, shape and depth, which were often covered with large sloughs. There was often found the lesions described by Morehead [46] as submucous suppuration. He speaks of preserved preparations of a colon which had been brought from China as pre-

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senting the appearance as of bran scattered over the surface, and says that he himself never saw such cases in Secunderabad, nor are there any such preserved in the Netley Museum. Fayrer[49] devotes most of his work to the description of the clinical features of the disease. His anatomical description of the lesions is very poor, and does not seem to rest on personal observations. He considers the disease as a unit, and the various forms described to be simply phases of the same process.

The observations of the French authors, made mainly in Algiers, are very similar to those of the English in India. The principal of these works is the classic and oft-quoted work of Dutroulau.[50] He calls attention to the similarity of the lesions in endemic dysentery. They may be described in two words—sloughing and ulceration. The lesions vary in intensity in the different cases. He describes as typical ulcers, those which are deeply excavated and with raised edges, and says that they are often so numerous that the mucous membrane looks as though a charge of shot had been fired into it. In some cases, there is a layer of pus between the mucous and muscular coat, resulting from the destruction of the submucosa. The mucous membrane is bossy and œdematous; it may become completely disorganized and changed into a greyish mass. There may be loose lamellæ of it, only attached by a few fibres to the subjacent tissue. The ulcers may extend to the muscular, and even to the serous coats. They always result from the casting off of a slough, and in some places, these sloughs are partly or wholly left in place. In one case, he describes the lesions as follows: "In the descending colon a peculiar condition is found; there are circumscribed, rounded elevations, as if an air bubble had formed between the mucosa and the submucosa. When these elevations are cut into, one can raise from them with a scalpel a small, round translucent body of gelatinous consistency and irregular shape, having approximately the size and appearance of the crystalline lens. On some of the prominences, one can see a yellow point, which gives exit to pus when incised." He denies the presence of a diphtheritic membrane in endemic dysentery, and affirms that the apparent membranes which are formed consist of exfoliations of the mucous membrane and detritus from surrounding ulcers. In some cases of acute dysentery, rapidly fatal from abscess of the liver, there may be only lividity, discoloration and infiltration

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of the mucosa. He is unable to say what structures of the mucous membrane are first attacked, for in his experience, the necrosis was so complete and usually so rapid, that it appeared to him that all the structures were affected simultaneously. The process is nearly always limited to the large intestine; but when it extends a short distance above the valve, the lesions are essentially different in nature and appearance from those observed in the large intestine apart from liver abscesses. In all the recorded autopsies to which he had access, and in his own cases, there was some alteration of the liver noted. In acute cases, the liver is hypertrophied, and of a reddish-brown color, often soft, and easily torn. In chronic dysentery, on the contrary, there is almost always some atrophy.

Berenger Féraud [61] divides dysentery into three forms—sporadic, endemic and epidemic dysentery. He regards dysentery everywhere as the same disease, and denies that any form of it is peculiar to the tropics. The dysentery of the tropics has the same character as that met with in Europe, the only difference in the tropics being in the frequency and severity. In the tropics, it is continually present, but in other regions, only during certain months of the year. He has seen the same forms in Senegal, and on the North Sea, and in Norway. The chronic dysentery succeeds the acute. This development of the acute into the chronic forms, is much more common in the tropics than in Europe, because the conditions for recovery are not so good. In the tropics, the chronic form either succeeds the acute directly, or it comes on after several attacks, and is established by insensible gradations. His views of the pathological anatomy of the disease are based on the study of 745 autopsies, made in Senegal, Martinique, or in the hospital Saint Mandrier, by himself or comrades, in the navy. No microscopical examinations were made of the lesions. The lesions which he finds agree with those described by Haspel. He considers all the different characters which the lesions present to be either due to different stages of the same process, or to different sorts of inflammation. Like most other observers of the tropical dysentery, his attention has been drawn to the character of the very small ulcers, which rather represent the opening of abscesses in the submucosa. The mucous membrane, he says, is often gangrenous over a considerable area; it can be rubbed away, leaving the muscularis perfectly bare. It is often elevated and supported by a

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layer, or small mass of pus. In the chronic form, the thickening and induration of the intestine is the most prominent feature. In spite of the fact that the author regards all forms of dysentery as of the same character, he makes no mention of the diphtheritic form, except to consider the question whether the large sloughs found floating in the intestine are the remains of tissue, or a diphtheritic exudation. It is obvious that among his 745 autopsies, there were very few cases of diphtheritic colitis. He considers the liver to be more or less affected in all cases of dysentery. In 411 autopsies of dysentery in Senegal, the liver was hypertrophied, softened or congested in 123; there was abscess in 143; discolored in 29; atrophic or cirrhotic in 18; and in 98 cases no changes could be detected by the naked eye.

Haspel,[52] in the third and fourth groups of his classification, has very well described the amœbæ dysentery. In the third group, the cellular tissue which separates the different coats of the intestine, is thickened and infiltrated with serous fluid, blood or pus. Projections are raised on the mucous surface by these accumulations beneath, forming irregular vesicles, flattened at the apex, and sometimes ulcerated. When this suppuration of the submucous tissue has invaded a large area of the intestine, the tissue above is detached in large masses, sometimes comprising the entire circumference of the intestine. In the fourth form of Haspel, large gangrenous masses of variable size and depth are found, which, in some cases, occupy one-third of the surface of the large intestine.

Gely [53] has observed various forms of dysentery, and considers the lesions as an alteration of the entire intestinal wall, not limited to the mucosa. There is engorgement of the follicles and the submucous cellular tissue, as is shown by the infiltration and thickening. He describes, under the name of "mammillated hypertrophy," the condition in which the small, tumor-like masses are formed on the mucous membrane. In the second form, the chief lesion is ulceration of the follicles; in the third, the formation of a false membrane; and in the fourth, the thinning and softening of the intestine.

The descriptions of dysentery given by Cambay, have chief reference to the clinical features of the disease. Kelsch and Kiener, [55, 56, 57, 58.] in their numerous publications on the subject of dysentery and abscess of the liver in the tropics, have described at length the dysenteric lesions with their pathological histology. In his first

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publication,[67] Kelsch speaks of the differences between the results of his investigations made in Val de Grace, and those of Cornil in Paris. He thinks, however, that the pathological process is the same in both the tropical and European dysenteries, and the differences are only due to the greater severity of the cases. The chief distinction between the chronic dysentery of the tropics and that of Europe, is that the first is a chronic disease which has passed through an acute stage, while the tropical dysentery is chronic in its inception. The first observation of Kelsch was on a case of the chronic dysentery of Cochin China. The case, a soldier, was attacked with dysentery one month after arrival in Cochin China. At first there was considerable abdominal pain, and the stools were lightly tinged with blood. Afterwards, the disease took the form of an ordinary chronic, rebellious diarrhœa, with liquid, yellowish stools, numbering 5–10 daily, and without a trace of blood. He returned to France and spent three months with his family, having 5–6 stools daily. He came into the hospital of Val de Grace in January. He was somewhat emaciated, and had 6 thin, vitreous stools daily, which were often streaked with blood. Gradually he became worse, and died on the 21st of March, the disease having lasted over one year. At the autopsy, most extensive ulceration, and in addition to this numerous small tumors, as large as a pin's head or a pea, filled with transparent mucus, were found. Kelsch refers these to the solitary follicles, and says that in addition to these changes in the follicles, there is constantly found thickening of the submucosa. The cells of the lymphatics are swollen, and there is much cellular infiltration around both the blood and lymphatic vessels. He does not agree with Gely, Thomas and Colin Chisholm, as to the presence of diphtheritic exudation. The principal lesions he groups under four forms:

1st. Redness, swelling and mammillated condition of the mucosa, and more or less swelling of the follicles.

2nd. Elimination of the follicles en masse.

3rd. Elimination of more or less extensive fragments of the mucosa, which can always be recognized as such from the presence of Lieberkühn's glands.

4th. More or less extensive ulceration following all these changes.

Much stress is laid on the lesions of the follicles where the ulcers are first formed. When once formed, however, they extend slowly, and

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enlarge in depth by a different process from that which produces them. The mucous membrane at the edge of the ulcer and the submucous tissue, are gradually destroyed. He thinks that the chief element in producing the necrosis and ulceration is the small cell infiltration around the vessels occasioning an obstruction to the circulation.

All of these lesions are described more in detail, with some additions, in the last publication of Kelsch and Kiener.[58] They call attention to the enlargement of our knowledge of dysentery, which close study of the vast territory of tropical dysentery has afforded. The tropical form differs from that in the temperate regions in the greater severity of the lesions, and in the phlegmonous and gangrenous form. In these, there is destruction of the entire mucous membrane by purulent infiltration, and the destroyed tissues are found as shreds or masses in the stools. They divide the lesions into two forms—the ulcerous and the gangrenous,—which correspond to two distinct forms of necrosis of the tissue—dry necrosis and gangrene. The first is a pure diphtheritic form. The mucous membrane is covered with smaller and larger necrotic and diphtheritic areas. In the mucous membrane there are small elevations from the size of a grain of wheat to that of a pea, with a slight necrotic area often tinged with bile on the surface. Beneath these there is cellular infiltration extending as a button into the submucosa. In this, three zones can be recognized—a central area of total necrosis, a middle one in which the elements, though necrotic, are still preserved, and an outer one of oedema and fibrinous infiltration. In these lesions, the follicles do not play any part. The process is fundamentally a necrosis of the mucous membrane, gradually extending into the submucosa. In the second stage, the necrotic mass is thrown out, and an ulcer is formed. Fistulous tracts may also be formed between neighboring ulcers, from the joining together of the infiltrated areas. The leucocytes accumulate in considerable numbers on the surface of the ulcers, and especially in the irregularities. They undergo a vitreous transformation, and form a reticular pseudo-membrane, adherent to the walls, resembling a diphtheritic membrane. This is only formed in places where the leucocytes can accumulate. It is not present on the free surface, probably because the leucocytes are swept away by the fæces. In the second, or gangrenous form, the lesions are more diffuse, and vary in intensity with the activity of the process. The intestine is generally

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contracted, and the peritoneal vessels are injected. The lesions occupy the entire surface of the colon, and often extend into the ileum. The surface has an irregular, mammillated appearance from the elevations and depressions. The ulcers and dry necrotic areas are larger and more numerous than in the first case. The submucosa is œdematous; its vessels are injected, and masses of fibrin are often found in it. The follicles take no part in the process. The muscular cells in the part affected are enlarged, and present a vitreous, refractive appearance.

In the gangrenous form, the lesions differ in some respects from the preceding. The typical primitive lesion in this is the formation of gangrenous sloughs. These always form a prominent plaque on the surface of the intestine; they are soft and humid, and rest upon an œdematous base. The sloughs vary in depth, and may invade the submucous and muscular tissue; they extend from point to point, until an area as large as the hand may be affected. The sloughs are usually detached without an eliminating inflammation. The intestine is friable and easily tears on extraction. The intestinal contents are of a liquid brownish color, often mixed with blood, and have a gangrenous odor. The mucous membrane between the ulcers is thickened and œdematous. The authors claim that all the lesions of dysentery may be produced in dogs by the injection of ammonia into the rectum.

Gayme,[59] Bougarel,[60] Julien,[61] have described the lesions found in the dysentery of Cochin China. Most of the lesions described by them can be referred to the amœbæ form.

Catteloup's [62] description of dysentery in the north of Africa agrees in general with the forms described by the other French authors.

Basch [63] has investigated the lesions in dysentery in material which was brought from Puebla, Mexico. He describes the thickening of the submucosa, the swelling of the connective tissue cells, and the bacteria which he found in and beneath the ulcers. It is difficult, from his descriptions, to say whether they have reference to the amœbæ form or not.

Coming now to the dysentery of Europe, one of the best articles on the subject is that of Vogt.[64] His descriptions of the lesions are very clear and objective. He divides the lesions into the catarrhal, croupous and gangrenous forms, and considers that all these are different types of one and the same disease. Most of his cases were acute and derived from an epidemic, and were evidently most of them

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cases of diphtheritic colitis. He is unable to understand the lesions which Annesley has described as occurring in the submucosa. He quotes, in the same connection, Becker,[65] who has described this condition much more fully and clearly. (We were unable to obtain this article of Becker's, which must form a valuable addition to our knowledge of the pathological anatomy of dysentery, and only quote from Vogt.) Becker describes an exudation between the mucous membrane and the muscularis, which consists microscopically of amorphous granules, six or eight times the size of a red corpuscle (swollen connective tissue cells or amœbæ ?). In its different forms, it appears as a diffuse laminated mass, or in oval, circumscribed plates 6–8 centimetres in diameter, or sometimes as lenticular or round masses. This exudation softens in the middle, and the softening extends to the periphery. The mucous membrane over it becomes necrotic and is cast off with the exudation. Vogt has never seen this exudation in the submucous tissue, and believes that it was only a different form of the exudation on the surface. He asks: Has perchance the idea that the dysentery has its origin in the submucous tissue influenced the observations of Becker, or is the Indian dysentery different from ours?

An excellent description of dysentery is given by Finger.[66] He divides the lesions into two forms: 1st. Intestinal catarrh, which has for its especial feature inflammation of the follicles. 2nd. Croupous inflammation of the intestinal mucous membrane. His description of the affection of the follicles corresponds very closely with what we have observed in the amœbæ form of dysentery, and with the descriptions of the disease in India and in the north of Africa. In this form, the mucous membrane is reddened, very loose and softened. The follicles are enlarged, and often appear as vesicles containing gelatinous mucous contents, and surrounded by a crown of injected vessels. On slight pressure, a glossy transparent mucus, or in some cases an opaque, thick purulent fluid can be squeezed from the follicles. Ulcers are formed from these follicles. They are at first solitary, and then, by the destruction of the intervening mucous membrane, they join together and form very large ulcers, whose edges are lax, villous and often swollen and suffused with blood. There is continual undermining of the edge of the mucous membrane around these. By the continual joining of these edges, large, irregular, ulcerated sur-

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faces are formed, which are joined together by loose bridge-like portions of the mucous membrane. When this follicular affection lasts a long time, which it is especially apt to do in children and in weak, anæmic persons, the intestine becomes much thickened. Both the mucosa and the submucosa become thickened and infiltrated with serum. The mucous membrane was sometimes pale, as though washed out, or of a dark greyish or brownish color, and often elevated in the form of yellow or grey ridges and elevations which were infiltrated with serum. In none of the cases were there abscesses of the liver. The epidemic described by Finger was frightfully fatal—107 deaths in 322 cases.

Moracchi [67] gives a somewhat similar account of the lesions. He says in the slightest cases there is a simple catarrh of the large intestine. The mucous membrane is swollen and is covered with a dirty-white, greyish-red or greyish-yellow fluid. In severer cases, the mucous membrane is softened, and with the bloody exudation, forms a more or less coagulated substance, which is easily scraped off. There are numerous elevations of the surface, which are caused by serous infiltration of the submucosa. These elevations are not over the entire surface, but where the affection is more intense. In some places the submucosa is generally thickened. In the further course of the disease, the elevations become higher, the inner surface is uneven and hilly, the submucous infiltration becomes purulent, and the mucous membrane appears dark brown or greyish-green. In the highest degree, the mucous membrane over large areas becomes necrotic. There are purulent and serous accumulations under the mucous membrane, and the muscular coat is pale and easily torn. The mesentery and mesorectum are often œdematous and gelatinous. The liver is enlarged, and abscesses are sometimes found; but he does not say in what proportion of the cases.

Cheyne [68] gives an account of the dysentery which prevailed among the troops and people of Ireland in 1818. In this, one of the earliest descriptions of diphtheritic dysentery is given. We were unable to obtain the still earlier publication of Osborn on the same subject. Cheyne divides the cases into two classes, according to whether the intestine was or was not thickened. In the second class, there are five conditions, referable to the condition of the mucous membrane. 1st. The mucous membrane is very vascular, without ulceration. 2nd.

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It is covered with coagulated lymph. 3rd. It is abraded, the epidermis only being removed. 4th. It is ulcerated, with intervening healthy portions. 5th. It is partly ulcerated and partly covered with lymph. In the first class, with thickened intestine, there were four conditions. 1st. the mucous membrane is simply abraded, and the intervening portions are healthy. 2nd. It is rugous and ulcerated. 3rd. It is ulcerated and hanging in shreds from the ulcers. 4th. It is partly ulcerated and partly removed, exposing the muscular coat. In a good many dissections there were numerous cavities large enough to admit the head of a pin, vascular in the centre and with elevated overhanging edges. He considers these to be enlarged mucous glands, and, in advanced stages, they were either ulcerated or connected with a cyst which secreted a gelatinous material. In four cases, abscesses were found in the liver, and in a considerable number of cases the liver was enlarged and congested. In the cases which he gives, there is a great deal of difference between the lesions in the acute and chronic cases, the acute being nearly always the diphtheritic form. The cases with abscess of the liver were all subacute or chronic. In two cases death took place at the end of the sixth week; in one, at the end of the eighth week, and in the other, at the end of the tenth. The lesions described in all these cases would agree well with the amœboid form.

Baly [69] gives an account of the epidemic of dysentery which prevailed in the Millbank prison. He considers the earliest changes the swelling of the solitary glands forming round prominences from the size of a millet seed to that of a shot. Later yellowish sloughs formed on the surface of these, which could be removed, leaving small ulcers. These lesions of the glands were seen especially in the acute cases. Some of the cases which he describes would seem to be almost certainly of the amœbæ form. One case is mentioned of a woman who had suffered for several weeks with dysentery of so mild a type that medical aid was summoned only two weeks before her death. The solitary follicles in the ascending and transverse colon were enlarged. Some of the glands along the prominences of the rugæ and the longitudinal bands were as large as a pea and open at their summits. From these a muciform fluid, in some instances transparent, in others opaque and yellowish, could be squeezed. Some of the largest of these, when laid open, would have received a horse bean into their cavity. In

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most of these cases, there was no thickening; but in others, which had passed into the chronic stage, both the mucous and submucous coats were thickened. At a later stage, the small sloughs enclosing the solitary glands are thrown off. The thin superficial layer of the surrounding mucous membrane loses its vitality and is thrown off. The mucous and submucous tissue are in most cases much thickened and tinged with blood and serum. The cavities which contained the sphacelated solitary glands appear as small, round ulcers, and give to the inner surface of the intestine the appearance of a worm-eaten piece of wood. The ulcers communicate with each other here and there in their deepest part, and sometimes large ulcers are formed by the destruction of the septa of mucous membrane which had formerly separated the small ulcers. The acute diphtheritic form could prove fatal in three days. In some of these acute cases, the mucous membrane formed an uninterrupted surface of disorganized tissue, of a dark, greenish color, rough and warty to the touch; it was of firm consistence, a half line in thickness, and subjacent to this there was a layer of dark coagulated blood. He does not think there is any difference anatomically between the dysenteric lesions which he has described and those found in the dysentery of the tropics.

Fagge[70] gives a very good description of the pathology of dysentery, the results of personal observation in Guy's Hospital. He divides the lesions into the catarrhal and diphtheritic forms. In the catarrhal forms, the mucous membrane shows lines and patches of a dark red color here and there, almost black. Both the mucous membrane and the submucous tissue are swollen and infiltrated with serum and pus cells. The solitary follicles are greatly increased in size, but this affection of the follicles forms only a part of the general inflammation of the intestine. Later the mucous membrane softens beneath the general cellular infiltration, and ulcers are formed. In severe cases, abscesses may also arise in the submucous tissue, and these may burrow, so that the undestroyed parts of the mucous membrane over them are detached in the form of bridges. When pressure is made upon these, pus may exude at several points. The inflammation may extend at one or more points through the muscular coat, and perhaps even penetrate the serous coat, so that perforation takes place. In spite of the differences in appearance between the lesions of these two forms, Fagge thinks that they merely represent differences in severity.

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Rokitansky [71, 72] divides dysentery into three forms: (*a*) In the mildest form the mucous membrane is cloudy, here and there reddened and injected, and covered with a pale greyish-red exudation which is easily removed on scraping it with the handle of a scalpel together with the mucous membrane beneath it. Here and there, the mucous membrane is either reduced to a very thin coating over the submucosa, or it is entirely gone, leaving this bare. The solitary glands are swollen, and the submucous tissue infiltrated. (*b*) In a higher degree of inflammation, the mucous membrane is covered with a dirty-white layer of necrotic epithelium, mixed with necrotic cells and mucus from the glands. It is reddened and injected, in various degrees swollen, rotten and easily torn. The submucous connective tissue is greatly infiltrated and elevated in the most affected portions. In addition, the mucous membrane is often covered with a croupous exudation, which is especially prominent on the extremities of the folds. On close examination the mucous membrane is perforated by numerous small openings which penetrate the submucous tissue. They are due to a partial or complete destruction of the solitary glands which have broken down in an acute suppurative process. The lymph glands in the meso-colon are swollen and softened. (*c*) In the highest grade, the mucous membrane over wide areas is converted into a slough, which often is passed out in tubular masses. The submucous tissue is infiltrated with serum; it is pale, and contains vessels filled with dark fluid blood. It later becomes infiltrated with pus, and often necrotic in large areas.

Cornil [73] gives a very complete account of the lesions studied by him in two cases of dysentery. He regarded the small nodules as formed by a suppuration of the solitary follicles, and described a downward growth of the epithelium from the mucous membrane until it often formed a complete layer around the wall.

Orth [74] gives a very complete account of the dysenteric lesions. He describes first the catarrhal condition, and refers to the constant involvement of the lymphatic apparatus, the lymph nodules and vessels in this. The changes in the vessels are often well marked and consist in swelling and proliferation of the endothelium. This is frequently so great that the lumen, for a considerable distance, is filled with the large epithelioid cells. When the swelling of the lymph follicles is very pronounced, the name "enteritis follicularis

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or nodularis" is given to the affection; but there is no sharp limit between the simple catarrhal and this form. This change in the lymph nodules consists in an increase in size of both the solitary and agminated glands, both those in the large and in the small intestine. There is also an enteritis follicularis apostematosa, which either may be combined with or appear independently of the hyperplastic form. The chief seat of this is in the large intestine, though the small may also be involved. In this form the lymph nodules swell to the size of a pea or larger; they become yellow and soft in the centre, and a pus cavity is formed, which is first small and then gradually involves the entire follicle. This breaks into the lumen of the intestine and a follicular ulcer results. Since the opening of the ulcer is very small, the edges of the mucous membrane overhang the cavity and float up where water is poured over it. In a chronic course of the disease remarkable changes take place in these follicular ulcers. The cavities become so filled up with glossy transparent mucus that they reach their former size, and the mucus projects into the intestine. This mucus comes from the epithelium at the edge which often grows down and forms a covering over the ulcer, in which gland ducts may develop. In other cases there is a more unfavorable course. The abscesses lie in the submucosa, and the suppuration may extend in this in all directions. In this way submucous abscesses arise, which may join with neighboring nodular abscesses and undermine the mucous membrane for a considerable distance. In some cases only small bridge-like masses of mucous membrane remain, and in others the mucous membrane hangs over the edges of the ulcer in long, ragged projections. The bases of these ulcers are generally formed by the muscular coat, which is either smooth and clear, or it shows irregularities and defects. The muscular coat can also be destroyed by suppuration, and the serosa laid bare.

The most exhaustive treatise on the subject of dysentery is that of Woodward.[75] The material for this was obtained from the cases which developed in the armies of the United States during the civil war. 878 cases with autopsies are reported, but the autopsy reports in most cases are so meagre, and the lesions so imperfectly described, that but little idea of the form of dysentery to which they refer can be obtained. Woodward adopts a classification, based entirely upon the clinical symptoms. He uses the word "dysentery" entirely in a

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clinical sense, and describes only those cases as dysentery in which tenesmus was present. The great value of Woodward's work is in the thorough analysis of all literature relating to the subject, and in the illustrations of the lesions. Although the number of autopsies was so large, but little of the material was available for microscopic examination. The material on which the microscopical examinations were made was derived almost exclusively from the hospitals in Washington and the vicinity. The work is illustrated by splendidly executed colored plates and photographs, both of entire specimens and slides. A considerable part of the book is devoted to the analysis of the cases of acute diarrhœa, the so-called "Chickahominy diarrhœa" of the armies in Virginia. The autopsies in these cases were made in the Philadelphia hospitals by Dr. Leidy, and these, together with those made by Dr. French in the army at the South, are the most important and the most carefully described autopsies in the book. In this acute diarrhœa, the chief lesions described are hyperæmia of the entire intestinal tract, with swelling of the lymphatic follicles. In some of the cases of intense congestion, there was sometimes slight erosion of the mucous membrane. From the descriptions in some of the cases, there seems to have been some diphtheritic colitis, but it must have been very slight, and present in but few cases. Under the head of acute dysentery, he considers all the various forms of acute intestinal flux in which tenesmus is a prominent feature. The tenesmus being due to sufficient involvement of the descending colon and rectum to make expulsion of the fæces painful. He makes two divisions of this. In the first, the lesions are almost the same as those found in diarrhœa, but are more pronounced in the colon and rectum. In the second, there is diphtheritic inflammation. Follicular ulceration was frequently met with. The lesions similar to those found in diarrhœa were from the milder cases. He says that acute dysentery does not usually prove fatal until more serious lesions have developed in the intestine. The inflammation may be so severe as to produce ulceration beginning in the follicles, and all that he has seen impresses him with the belief that fatal cases of follicular colitis are more common than diphtheritic. "Follicular ulceration, attended by the well known concomitant thickening of the submucosa, was an exceedingly frequent lesion in the bodies of those who died of flux; but when a complete history of the case could be obtained, it usually

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proved that the patient had suffered from looseness of the bowels, either continually or at intervals, for several months. He considers the cases in which extensive sloughing was found as belonging with the diphtheritic form. The colored plate which he gives on page 454, seems to be a very good illustration of what we regard as the amœbæ form of dysentery. The patient from whom this was obtained was taken sick on August 11th, and died December 13th. Another case, with a careful autopsy by French, was sick from July to November 18th. In this case, an abscess was found in the anterior superior part of the right lobe of the liver containing 20 ounces of pus, which had the odor of sour milk." Of the intestinal lesions, French says: "The mucous membrane of the whole tract of the large intestine was of a mottled, livid, greenish color, stained here and there with darkened spots, and spread as if worm-eaten with ulcers of varying depth, some penetrating the mucous coat, others the muscular coat, and two through the serous coat of the bowel. In the lower half of the colon, the ulcers were so numerous that the intestine presented a worm-eaten appearance like tripe. Some of these had rough, ragged borders. In others, the borders and bottoms of the ulcers were smooth and glazed. Others were lined with opaque plastic lymph, which was tenaciously adherent. The shape of the ulcers, though very irregular, was mostly oval, the long diameter extending across the bowel." It seems certain that some of Woodward's cases, which he reckons among the diphtheritic forms, especially those in which the sloughing was well marked, belong to the amœbæ form of dysentery. He classes these cases which were all of long duration, among the acute forms, supposing they had had diarrhœa for some time which passed into acute dysentery. In his chronic dysentery, he makes two forms, in one of which there is simple chronic inflammation without ulceration, and in the other, the formation of follicular or other ulcers. Of the latter, he says the pathological picture is often complicated by the development of diphtheritic inflammation between the ulcers. Of the follicular ulcers, he says: "The follicular ulcer in the earliest stage may be recognized by the fact that it is seated at the apex of an enlarged follicle, where it appears as a tiny irregular excavation. At a later period the entire gland is destroyed by ulceration, and the ulcer appears as a little cavity in the submucosa, and communicates with the surface by a constricted orifice. Subsequently, the ulcer extends in the submucosa

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undermining the mucous membrane which perishes in proportion as its nutritive supply is cut off from beneath and the orifice enlarges with the ulcer. Still later, adjoining follicular ulcers coalesce forming large, irregular excavations, by means of which, in extreme cases, a large area of the mucous membrane of the colon is destroyed. These ulcers never present the beveled edges of those which result from diphtheritic sloughing, but always, unless complicated by this latter process, have overhanging edges, from the fact that they spread more rapidly in the submucous than in the mucous coat." In another place, he says: "Intestines invaded by follicular ulceration usually present a number of other ulcers not connected with the follicles. These appear first, as superficial erosions, by which more or less of the thickness of the mucous membrane is destroyed. Ultimately, however, they invade the submucous connective tissue. In the great majority of cases in this class, ninety-nine in all, the clinical history is that of an intestinal flux, of from two or more months duration." Two hundred and ninety-three of the cases he has grouped as undetermined cases, in which from the imperfection of the autopsy reports, it is impossible to say to which form they should be assigned. The lesions in these cases are variously described as "numerous large ulcers," "colon extensively ulcerated," "a mass of ulcerations," "much ulcerated throughout." In five per cent. of Woodward's cases of follicular ulceration, abscess of the liver was found. But only eleven abscesses were found in the doubtful cases. It is certain that many of the cases described as "follicular colitis" were really the amœba form; this is evident both from the descriptions and from the very carefully made drawings and photographs which accompany them. Some of the cases of diphtheritic dysentery or rather the cases which he has, we think, wrongly included under this head and in which sloughing was a prominent feature, and some of the undetermined cases also, come under this head. We have given so much space to this book of Woodward because it is the most complete description which we have of the dysentery prevalent in the United States. It is greatly to be regretted that the imperfect descriptions of the lesions found at the autopsies impair the value of the work.

In connection with Woodward's book, we have examined the colon specimens in the Army Medical Museum in Washington. They represent much of the material which Woodward worked on,

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and many of them are represented among his illustrations. We examined all the specimens; picked out those which seemed to represent the amœba form, and on consulting the histories of the cases, found in each one a clinical history which corresponded to this diagnosis. There are 14 such specimens coming from 11 autopsies. Some of them are very typical, as 7811, 7812, 7813, which all came from the same case. The description of this case (a soldier in the Army of the Potomac), says: "The colon in this case was somewhat thickened, and presented numerous excavated ulcers, many of which penetrated to the muscularis and some to the peritoneal coat. These ulcers appear to have extended by burrowing in the submucous connective tissue below the true follicular layer. As a consequence, the latter hangs out in more or less extensive fringes, which are especially remarkable in one of the specimens. The patient was admitted to Douglas Hospital, with chronic diarrhœa; said he had been sick for a long time; did not know how long, but thought it was more than two months." These specimens are nearly all from the hospitals around Washington, which received the patients from the Army of the Potomac, or they are from the autopsies made by French in Vicksburg. Of course, it could not be said from this that this form of dysentery was most prevalent among the troops in Virginia and in Mississippi, but the material from most other places was not available. We have also examined the lesions of dysentery which are represented in the principal atlases of pathological anatomy. In these most of the illustrations are of diphtheritic inflammation. It is impossible to say from any of them anything of the character of the lesions. The only plate found which exhibits a typical picture of the amœba form of dysentery is one in the "Museum preparations of the Army Medical Department at Chatham."[76] In this (Fig. 1, Plate 14), the whole length of the colon is represented. It is very much thickened, contains numerous small ulcers, and there are large areas of the muscularis laid bare from sloughing. Nothing is said of the specimen but that it came from a case of chronic dysentery. Of these illustrated works, we have examined that of Hope,[76] Carswell,[78] Lebert,[79] Cruveilhier,[80] Albers,[81] Gluge,[82] Baille.[83]

We were able to find little which would show the distribution of the disease in America from the medical journals which are published here. Cases which appear to belong to it are reported in the

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medical journals all over the country. It would be too much to review these single publications. The disease would seem to be generally distributed. More cases are reported in the journals of the Northern and Middle States than in the Southern. In the Southern journals there are numerous articles on the subject of dysentery, but they relate almost solely to the treatment.

There is no very extensive literature on the anatomical changes in liver abscess. Most of the descriptions found in the text books on pathology do not refer to the form of abscess under consideration. Kelsch and Kiener[55, 58] have studied these abscesses in Algiers. They ascribe to these abscesses a typical character, and divide them into two forms—the phlegmonous or diphtheritic abscess, and the fibrous. In the first form, there is a rapid advance of the abscess. The tissue first becomes filled with pus cells; the whole tissue then softens; the cells become necrotic, and the walls become covered with embryonic granulation tissue, which, instead of going on to the formation of fibrous tissue, also becomes necrotic and converted into a diphtheritic membrane. Such abscesses have ragged walls; often there are long necrotic digitations projecting into the abscess, and the abscess extends irregularly into the surrounding liver tissue. Frequently there are small abscesses in the immediate neighborhood of the large. The second form is the fibrous abscess. In this, also, necrosis is the dominant lesion; but in place of the formation of a diphtheritic membrane around the abscess, there is a formation of fibrous tissue.

Kartulis[15] thinks that dysenteric abscesses are caused by amœbæ which enter the liver by means of the portal vein. The lesions which they produce are caused by their active movements, and by the bacteria which they bring with them. In the liver, they cause the capillaries to rupture, and hemorrhage into the parenchyma to take place. In the abscesses, they are most numerous in the margins, where they are almost confined to the capillaries. In addition to the large abscesses, he mentions that there are small necrotic abscesses, which are due to thrombi, and in which he has never found amœbæ or bacteria. It was in these small abscesses that we found the bacteria most numerous.

It will not be necessary to give any more of the literature of the subject. From what we have gone over, it will be seen that no single form of dysentery is peculiar to the tropics, and entitled to be regarded

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as tropical dysentery. The same lesions which have been described by English writers in India, and French in Algiers, are also described by English, Irish and German observers; and by Woodward and ourselves in this country. One thing seems certain—that the lesions in most of the epidemics of dysentery are those of diphtheritic colitis. It was this form which Vogt saw in his cases; it formed the bulk of the cases described by Baly in England, and Cheyne in Ireland; it was the most common form in the numerous cases which occurred in the Bay View Almshouse, at Baltimore, from 1880–1886. Among these Bay View cases, there were some which certainly belonged to the amœba form.

Diphtheritic colitis is a distinct affection, and the lesions produced by the amœbæ have nothing to do with this. In some of the descriptions, it seemed probable that the two forms coexisted. In one of the cases reported by us, in which amœbæ were found on sections of the tissues, but were not found during life or in the examinations made at the autopsy, there was a typical diphtheritic enteritis confined to the small intestine. There is another form of dysentery in which the lesions are different from both the diphtheritic and the amœba forms of the disease. The following case, for which I am indebted to Dr. Brune, is an excellent example of this:

The patient, a man aet. 63, died at St. Joseph's Hospital from carcinoma of the stomach, with metastases. He had dysentery for two weeks before death. He complained of much pain; the stools were fluid and contained no blood; there were numerous ulcers in the colon, the descending portion and rectum being most affected. The ulcers were generally superficial, many of them confined to the mucous membrane. Others were deeper; but none of them extended to the muscular coat. Some of the ulcers were perfectly round; others of very irregular shape, produced by the union of adjoining ulcers. The edges were smooth and never undermined. In the middle of some of the ulcers there were portions of intact mucous membrane, forming small islands, projecting above the surface of the ulcer. The intestine was not thickened, except about the largest and deepest ulcers, where there was some thickening of the submucous coat. The mucous membrane generally was pale and soft. There were some slightly elevated portions of it which were almost opaque and softer than the rest. The tissue was hardened in alcohol, and sections were made through all parts of it.

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On examination of the mucous membrane at a slight distance from the ulcers, numerous changes were found. The more superficial portions were broken down into a granular-looking mass, in which only the remains of cells were found. Deeper down there was a diffuse infiltration with pus cells, most marked just above the muscularis mucosa, and the glands of Lieberkühn showed various stages of disorganization. In some cases, they were dilated into thin-walled cysts, filled with pus cells; in others, the cells were separated from one another, forming irregular masses of epithelium, the cells generally swollen, and with indistinct outlines. There was a general increase in the lymphoid cells; the solitary follicles were somewhat enlarged and continuous with lymphoid cells in the mucous membrane. In the mucous membrane, corresponding to the whitish areas, there was a diffuse suppuration of the tissue. The glands were broken down, their confines lost, and they were represented by irregular masses of epithelium, surrounded by pus cells. There was a great deal of nuclear detritus coming from the cell destruction.

The ulceration extended through the muscularis mucosa—sometimes in only very small areas; at others, the entire ulcer was seated in the submucosa. In these cases, the submucous tissue was somewhat thickened, and the edge of the ulcer marked by granulation and pus cells. The pus cells extended deeply into the submucous tissue, sometimes diffusely infiltrating it; at others, collecting in groups. Around the vessels everywhere there were dense accumulations of cells, chiefly granulation cells, swollen connective tissue corpuscles, and pus cells. The most marked feature was the enormous swelling of the connective tissue cells in the neighborhood of the ulcers. They were converted into large, irregularly-shaped masses, with a large vesicular nucleus. Scattered through the swollen connective tissues there were numerous masses of fibrin. The vessels showed an increase in number and swelling of the endothelial cells; their walls were thickened, and there were numbers of pus cells both in the lumen and in the walls. In the submucosa, the cellular infiltration was very extensive just above the muscular coat, and the connective tissue septa between the muscular fibres were swollen and filled with cells. There was a great increase in the number and size of the plasma cells.

Bacteria were found, but they were not numerous. The forms met with were: micrococci, sometimes arranged in chains; short, thick

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bacilli and long bacilli, similar to those described in some of the intestinal ulcers in the amœba dysentery.

It is this form of dysentery which best conforms to the descriptions given under the head of catarrhal. It is essentially a suppurative inflammation of the mucous membrane, extending from this into the deeper tissue.

The intestinal lesions produced by the amœbæ in general conform to the lesions which have been described under the name of follicular dysentery. There is a form of follicular enteritis in which Peyer's patches and the solitary follicles, in both the large and small intestine, are involved. This was the lesion seen in the soldiers during the civil war, who died of the Chickahominy diarrhoea. We occasionally see similar cases here, but these do not proceed to ulceration. How many of the cases which are described as follicular ulceration, are really such? In how many cases the ulcers start in the follicles, we do not know. Nothing is more natural than upon seeing the enlarged follicles in certain cases, and in others the small nodular ulcers, to regard the latter as coming by gradual transition from the former. In the amœba form the ulceration does not begin in the follicles, and when these are affected it is only in common with the other tissues in the submucous coat. While we believe that the ulceration in the intestine is more or less characteristic, still our recognition of the amœba dysentery as a definite disease, is not founded on the lesions of the intestine alone, but on the character of the lesions in the liver and lungs. More than anything else, the character of the pathological processes in the various organs, as seen in the histological study of the lesions, leads us to recognize the peculiar character of the process which determines the lesions. The ulcers in the intestine often have their gross characteristics modified by the presence of other organisms than the amœba, giving a mixed character to the lesions. We know how difficult it is to determine the character of an ulcer, even when it comes to distinguishing between two ulcers usually so characteristic as the typhoid and tubercular ulcer of the intestine. We frequently have to use other criteria than the microscopic appearance of a single ulcer, as the tubercles on the peritoneum over the ulcer, etc. Still more difficult is the differentiation of a syphilitic and tuberculous ulcer upon a mucous membrane.

Abscess of the liver certainly is more frequent in this form of dysentery than in any other, though certainly not confined to this.

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With a view of determining the frequency of the association we have gone over the literature of abscess of the liver in the tropics and elsewhere. Some mention has been made of this in connection with our review of the literature of dysentery. We have found abscess of the liver in six of our autopsies. In any estimate as to the frequency of liver abscess we also have a difficulty, because the character of the abscess is not described with sufficient distinctness by most authors, and pyæmic abscesses are not clearly separated from the others. In these statistics, in reference to liver abscess and dysentery, we have made large use of the exhaustive article on the subject in Woodward's book. The subject of abscess of the liver has received the fullest treatment from the English physicians in India.

AUTHORS.	AUTOPSIES ON DYSENTERY.	LIVER ABSCESS.
Ballingall,[84]	35	4
Annesley,[42]	51	26
Waring,[85]	259	69
Eyre,[86]	118	27
Ranking,[87]	140	41
Moore,[88]	494	90
Macpherson,[89]	293	46
Chuckerbutty,[90]	39	3
	1429	306

From these statistics collected from every part of India, we see the enormous percentage of liver abscess there; about one case of liver abscess to every four and a half of dysentery. The percentage which Annesley gives is obviously too high, and is due to his having selected cases which especially illustrated the subject of hepatitis.

In Algiers, the proportion is almost as high as in India.

AUTHORS.	AUTOPSIES ON DYSENTERY.	LIVER ABSCESS.
Catteloup,[91]	240	47
Mouret,[92]	761	133
	1001	180

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180 cases out of 1001, or a proportion of one case of liver abscess to a little over five cases of dysentery. In Cochin China, Bougarel and Gayme, found liver abscesses in 13 out of 52 autopsies on dysenteries. Julien on the other hand, reports only 8 cases in 108 cases of dysentery.

The observations of DeCastro,[93] show that the disease is a very common complication of dysentery among the Europeans in Egypt, while Griesinger,[94] found not one case in 186 autopsies on dysentery in native Egyptians. The testimony of all observers is that it is much more frequent among the Europeans in the tropics than among the natives. When we turn to the statistics on this subject in Europe and America, the infrequency of liver abscess as compared with the tropics is very striking. Rokitansky in his essay on the dysenteric process, declared he had never encountered this complication, nor did Finger find an example of it in the 231 autopsies he made in Prague. Baly did not encounter it in the hundreds of autopsies he made in the Millbank prison. Lyons, whose observations were made on the soldiers in the Crimean War, only found one liver abscess in 51 autopsies on dysentery. Cheyne found it in 5 out of 30 autopsies made on dysentery in Ireland, in 1818. The only statistics which we have of the relation between the two affections are those of Woodward, who had 5 cases of it in 90 cases of his follicular dysentery; and 11 cases in 293 unclassed cases. Fox,[95] found it at St. Louis, 5 times in 30 autopsies on chronic diarrhoea and dysentery.

This much greater frequency of the complication of liver abscess in the tropics than in Europe and America, in what we must regard as the same disease, is inexplicable. Certainly many of the cases described by Finger we would regard as typical cases of amœba dysentery. One of us, during a course of study at the pathological laboratory at Prague, under Professor H. Chiari, became familiar with the lesions of dysentery as it existed there. It prevailed extensively at the time, and there was the greatest similarity between the lesions seen there and those of our own cases. Specimens brought from there, which are in our Museum now, could be used as typical specimens to illustrate amœba dysentery. Hlava has also found the amœba in 60 cases of partly sporadic, partly endemic dysentery. He says nothing about abscesses of the liver, and we presume that they were not present in his cases. The same was true in the dysentery

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of Vienna. We never saw in Europe a single abscess of the liver which could be regarded as due to the amœbæ. They were nearly all pyæmic abscesses spreading along the branches of the portal vein, such as Orth figures in his text book. In Egypt, where the amœba dysenteriæ was first discovered, and where dysentery ranks as among the most common diseases, abscess of the liver is certainly more rare than in either India or Algiers. On the other hand, Cheyne in his investigations in Ireland, found abscess of the liver in 5 out of 30 cases.

From all reports on the subject, we know how rarely liver abscess is found in England. When found there it is usually in sailors who have returned from a voyage to the tropics, or in people who have contracted dysentery in India. We have, however, found several cases reported of typical dysenteric abscess in people who have never been out of England. Dickenson[96] and Moore,[97] report typical cases. In both, the disease began with diarrhoea, which lasted several months; and at the autopsy extensive ulceration was found in the colon. In Dickenson's case there was one large abscess; in Moore's there were two. It is possible that the disease may be more virulent in certain countries, and there may be conditions which predispose to liver abscess. We cannot, however, learn that this is the case from the histories of the cases.

From all we can learn, hepatic abscess is certainly frequently met with in the tropics without any intestinal lesions. These abscesses have the same characters as those which accompany dysentery. We do not know whether amœbæ are present in these. If they all are produced by the amœbæ, it is possible that they enter the liver without producing any intestinal lesions. The matter is one which can only be cleared up by further research. We think it can be said, as the result of the work which has been done on the subject;—

1st. Amœbic dysentery is a form of dysentery which ætiologically, clinically and anatomically should be regarded as a distinct disease.

2d. We would consider that,

(*a*) The amœba dysenteriæ has been shown to be the causative agent from its constant presence in the stools and in the anatomical lesions, and from the inoculation experiments of Kartulis.

(*b*) Clinically the disease is characterized by the presence of amœbæ in the stools, which in addition present physical characters

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different from those seen in the stools of other forms of dysentery, as noted above; by a variable onset, course and duration of which the special features are periods of intermission alternating with exacerbations; and by a marked tendency to chronicity, with the production of a greater or less degree of anæmia.

(c) Anatomically the disease is characterized by the production of ulcers in the colon which generally differ from those found in any other forms of dysentery. The ulceration is produced by infiltration of the submucous tissue and necrosis of the overlying mucous membrane, the ulcer in consequence having the undermined form. Frequently in addition to the ulcers there is infiltration of the submucous tissue without ulceration. In all of these lesions unless complicated by the action of bacteria there is absence of the products of purulent inflammation.

3rd. Abscess of the liver with or without involvement of the lung is a frequent complication, much more so than in any other form of dysentery. The involvement of the lung may early follow hepatic involvement and be detected by the occurrence of amœbæ in the sputum before there is evidence of liver abscess. These abscesses differ in their anatomical features from those produced by other causes. The chief difference is found in the absence of purulent inflammation, the abscess being caused by necrosis, softening and liquefaction of the tissue. In these liver abscesses the amœbæ are not associated with any other organisms.

4th. The disease is widely distributed, and is found in most countries in Europe, in most parts of the United States and in the tropics everywhere.

5th. This is the form of dysentery which has been commonly called tropical dysentery.

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Erratum.—Page 396 in foot note instead of Lambert[3] read Lambl[3].

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DESCRIPTION OF PLATES.

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PLATE I.

Representations of sections through various forms of ulcers in the colon.

Figure 1.—Section of an elevated deeply excavated ulcer; at *f* there is an area of cellular infiltration at a slight distance from the ulcer. This extends into the mucous membrane and amœbæ were found at the edge of the infiltration. *a*, Mucous membrane; *b*, muscularis mucosa; *c*, submucosa; *d*, muscular coat; *e*, peritoneum. This lettering is the same for all the figures in this plate.

Fig. 2.—Section of an ulcer with both edges deeply excavated.

Fig. 3.—Section comprising a part of two ulcers, *g*, *g*, and a cross section, *f*, of an undermined passage in the submucosa.

Fig. 4.—Section showing extensive undermining and destruction of the mucous membrane. *f*, A small portion of mucous membrane adhering to the ulcer.

Fig. 5.—Section of a deep ulcer extending down to the muscular coat. The cellular infiltration, *g*, has extended through the muscular coat at *f*. There are numerous amœbæ at the edge of this infiltration.

Fig. 6.—Section of a deep ulcer at the base of which the muscular coat is also destroyed.

Fig. 7.—Section of excavated ulcer in the thickened submucosa. In this there are numerous foci of infiltration and softening.

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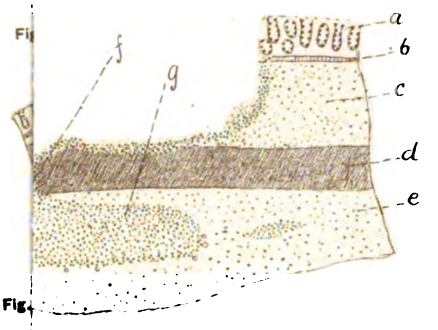
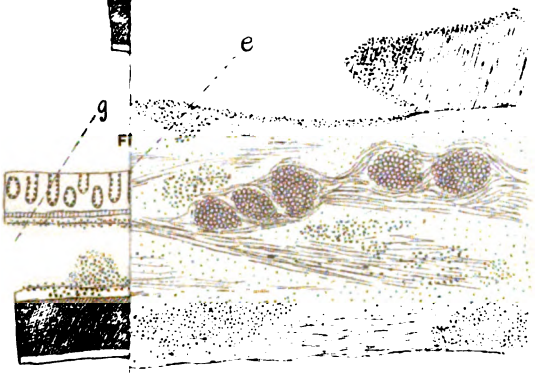
PLATE I.





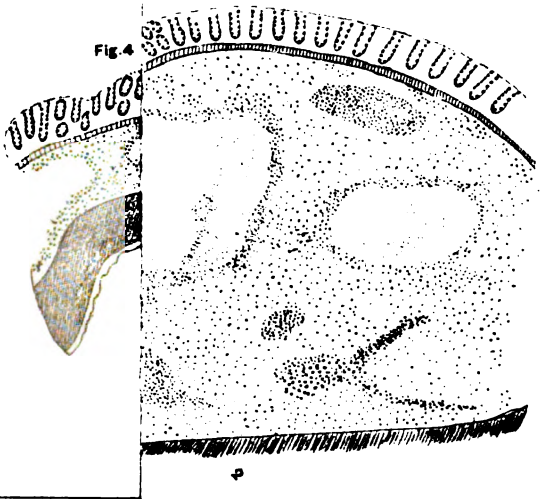


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PLATE II.

Fig. 1.—Section showing early stage in the formation of an ulcer. The submucosa is greatly thickened and œdematous. In the middle of the elevation it is infiltrated with cells and is undergoing softening. A small cavity has been formed which is continuous with a small softened area in the mucous membrane.

Fig. 2.—The hyaline reticulum formed in the bases of some of the ulcers.

Fig. 3.—Section near an ulcer showing the undermined tract in the submucosa. The growth of the epithelium has extended down from the surface; *a*, covers the roof of the excavation; *b*, contents consisting of granulation and pus cells, amœbæ and swollen connective tissue cells.

Fig. 4.—Section of the edge of an ulcer showing the growth of the epithelium. It extends downward at the sides and partially covers the floor of the ulcer. *a*, New formation of glands.

Fig. 5.—Section showing portion of a bundle of muscle in the base of an ulcer. The dark portions represent the remains of the muscular fibres. In the clear spaces these have disappeared or have become converted into a clear homogeneous material.

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PLATE II.

Fig.5

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PLATE III.

Fig. 1.—Section near the ulcer represented in Fig. 2, Plate 4, showing a portion of the thickened submucosa, the muscularis mucosa and the lowest portion of the mucous membrane. *a*, *a*, Lymphatics just beneath the muscularis mucosa containing amœbæ. In one of the vessels two amœbæ are seen apparently in the act of passing from this into the tissue. The amœbæ are also in the muscularis mucosa and in the mucous membrane.

Fig. 2.—From the same section near the muscular coat, showing the amœbæ near a ganglion, *d*. The nerve cells are represented in order to show the comparative size of the amœbæ. These are in the lymph spaces of the tissue at *a*, and in a lymphatic vessel at *b*; *c*, fibrin.

Fig. 3.—Section showing lesions of the mucous membrane. A small mucous cyst containing amœbæ. They are also seen in the mucous membrane between the glands. *b*, Amœbæ.

Fig. 4.—A portion of the meso-colon where the ulceration has extended into this. There are great numbers of amœbæ in the tissue, which is mostly necrotic. *a*, Amœbæ; *b*, necrotic fat cells.

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PLATE III.

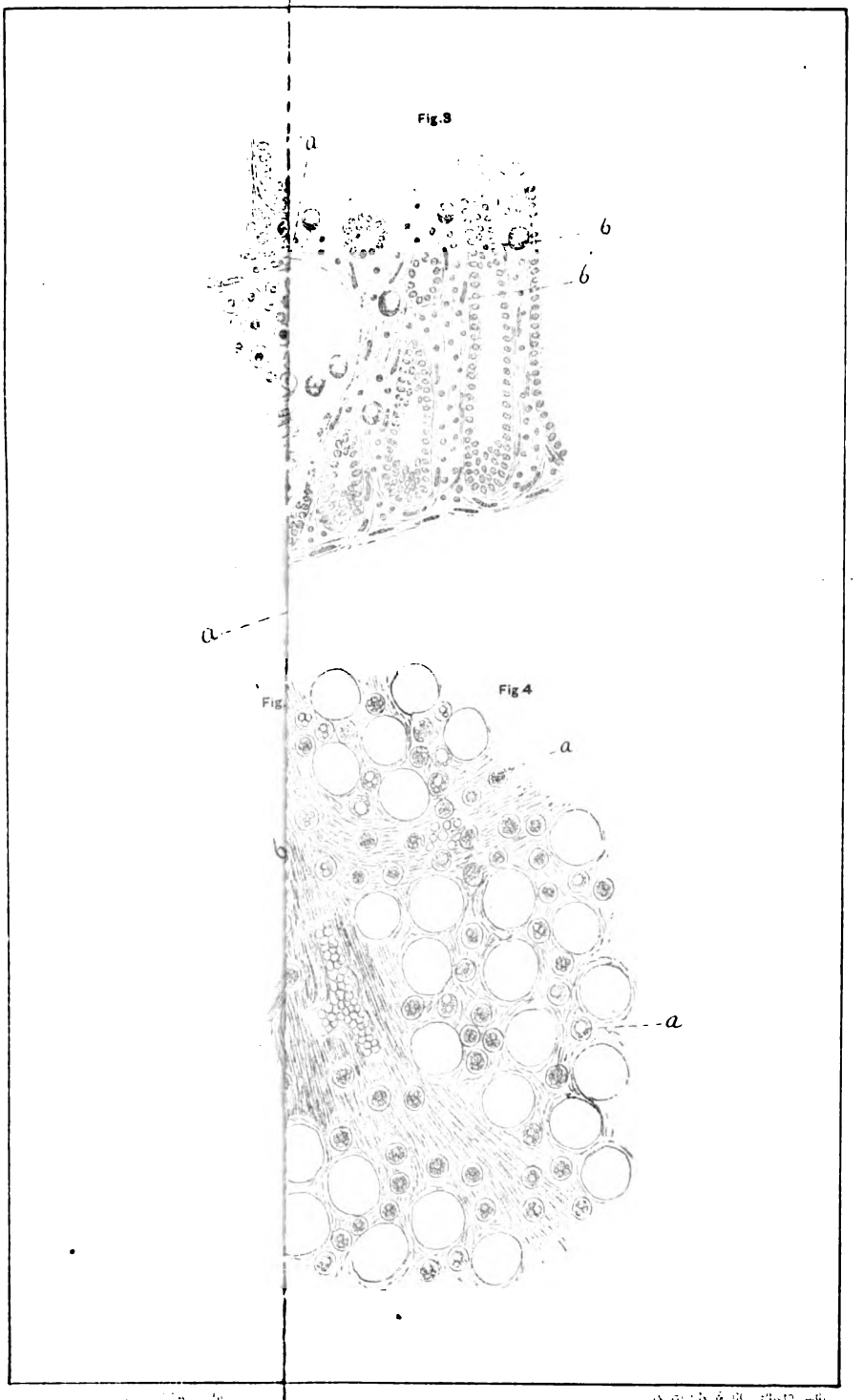

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PLATE IV.

Fig. 1.—Section showing excavated ulcer with purulent contents. The entire edge of the mucous membrane with the muscularis mucosa is turned in over the ulcer. *a*, Solitary follicle; *b*, bacteria in the purulent contents of the ulcer; *c*, pus; *d*, amœbæ.

Fig. 2.—Section showing slightly excavated ulcer which extends only a short distance into the submucosa. *a*, Solitary follicle; *b*, amœbæ.

Fig. 3.—Edge of small liver abscess. The liver tissue is necrotic and the nuclei do not stain. The cells are represented by trabeculae with greatly dilated capillaries. There are long necrotic cells in the contents of the abscess, together with numerous amœbæ. The amœbæ are also seen in the capillaries of the atrophic liver tissue and in the abscess contents. *a*, Liver tissue; *b*, necrotic liver cells; *c*, amœbæ.

Fig. 4.—Section showing necrotic liver tissue around the intralobular vein. The liver cells are separated from each other; they are partly preserved entire, partly broken up into small fragments, and lie loose between the capillaries. *a*, Intralobular vein; *b*, necrotic liver cells; *c*, fragments of the same. The dark-stained irregular bodies in the tissue are leucocytes.

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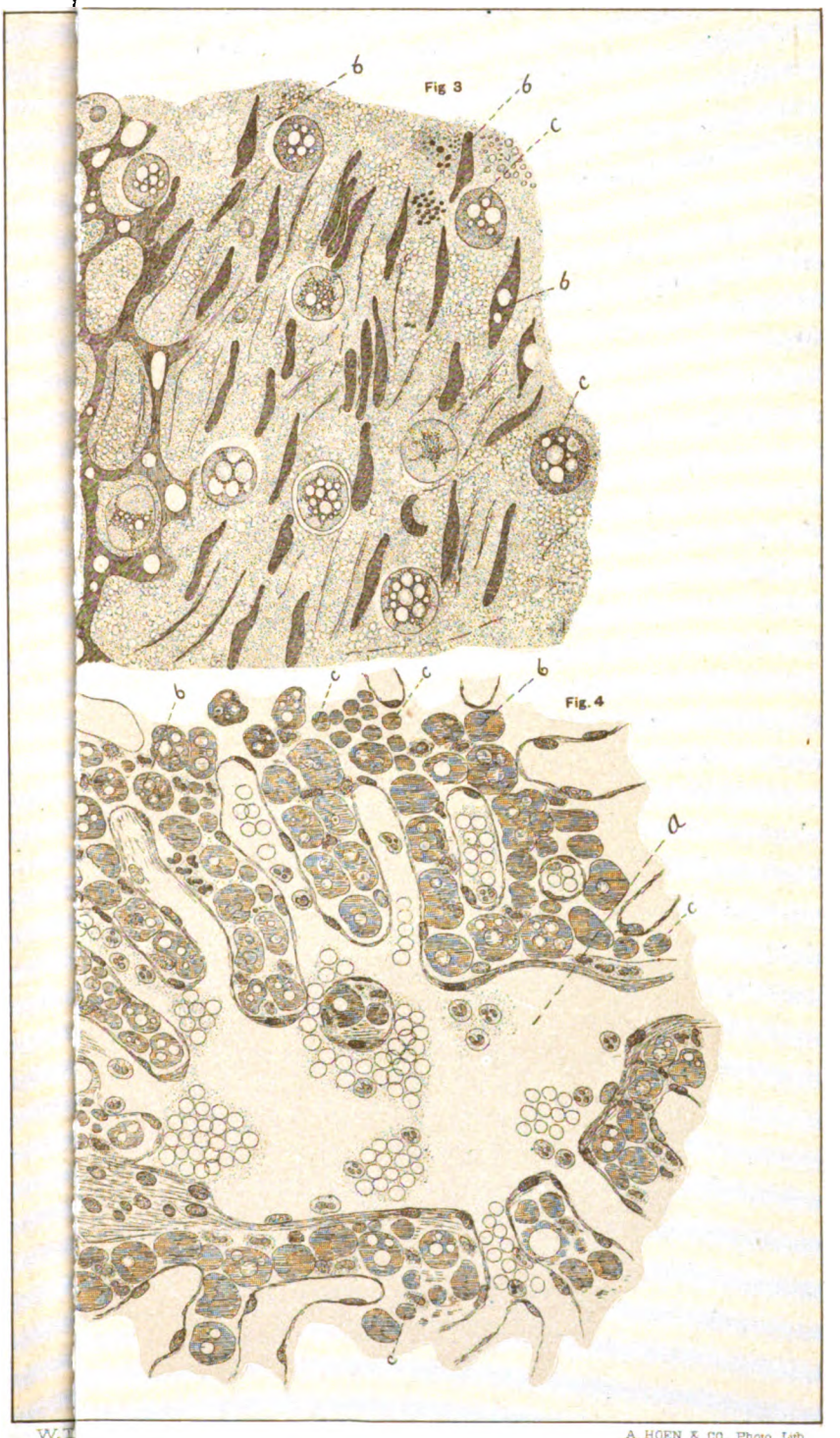

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PLATE V.

Section through a small abscess of the liver. In the abscess there are seen the remains of the portal tissue. The pale areas in the surrounding tissue are the necrotic liver cells around the centre of the lobules. The cells around the periphery of the lobules are preserved and their nuclei are stained. The abscess extends into the necrotic tissue, leaving small remains of liver tissue about the portal spaces, which form the solid masses in the abscess.

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PLATE V

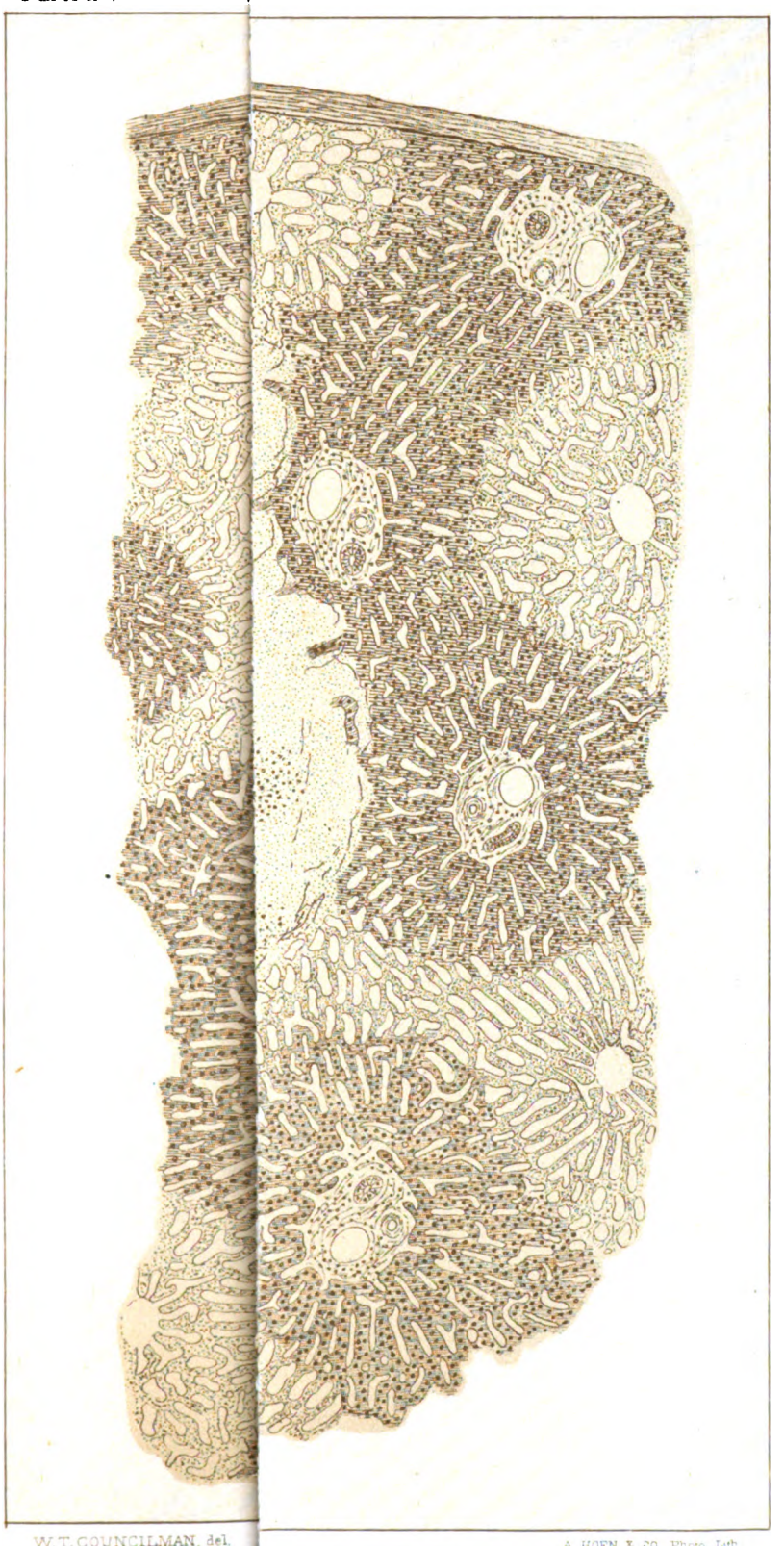

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PLATE VI.

Section through the fibrous wall of a liver abscess. *a*, Contents of the abscess; *b*, reticular and hyaline membrane; *c*, connective tissue; *d*, bile ducts; *e*, liver cells. There are numerous amœbæ, *f*, in the abscess contents and in the wall. In the last they are in spaces in the tissue, and in the blood vessels.

Fig. 2.—Section of lung adjoining an abscess. The alveoli are very small and irregular in shape; they are lined with cuboidal epithelium. The connective tissue is greatly increased in amount and extends into the alveoli. *f*, Bronchus containing fibrin and connective tissue; *e*, large swollen epithelioid cells in some of the alveoli; *c*, fibrin; *b*, connective tissue; *a*, alveoli.

Fig. 3.—Section showing bronchus near an abscess containing amœbæ in its lumen and in the thickened wall.

Fig. 4.—Section through the edge of an abscess of the lung. There is no fibrous wall to the abscess. There are remnants of lung tissue in the abscess contents. A larger mass of tissue in the middle of the figure is almost severed from its connection with the wall by the softening. There are numbers of amœbæ in the contents of the abscess and in the tissue at the edge. *a*, Edge of abscess; *b*, remains of lung tissue; *c*, amœbæ.

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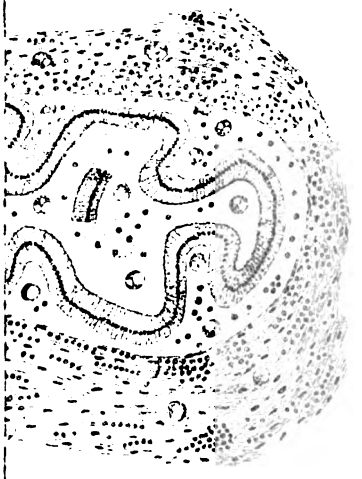
PLATE VI.

Fig. 3



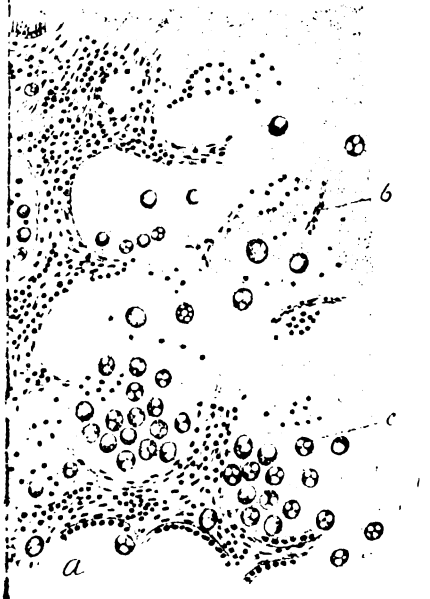


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PLATE VII.

The drawings in this plate were made from sections which were hardened in various ways. The coloring is as near to the specimens as possible. The amœbæ are all drawn larger than they appeared under the microscope. We are indebted to Dr. G. H. F. Nuttall for the very careful drawing.

Figs. 1, 2, 3.—Amœbæ from a section of intestine hardened in alcohol and stained with methylene blue. In Figs. 1 and 2 the granulation is very distinct. In Fig. 3 the dark staining at one end is seen, which frequently shows as a crescent under a lower power.

Figs. 4, 5, 6, 7, 8 and 15.—Amœbæ from sections of intestine and abscess of liver hardened in strong Müller's fluid and stained with hæmatoxylin and eosin. In Figs. 4, 6 and 8 the nucleus, *n*, is very evident. In Fig. 5 there is a large vacuole, possibly resulting from the rupture of several vacuoles. Figs. 7 and 15 are examples of amœbæ much smaller than the others.

Figs. 9, 10, 11, 12, 13 and 14 are from various tissues hardened in Flemming's solution. In Figs. 9, 11 and 12 the central mass has shrunken from the periphery. In Figs. 11 and 12 nuclear detritus in the form of small brightly staining rods is seen. In Figs. 13 and 14 are the radiate-like structures described in the text. In Figs. 10 and 13 are portions of the nuclei of included cells. In Fig. 10 there are included red blood corpuscles.

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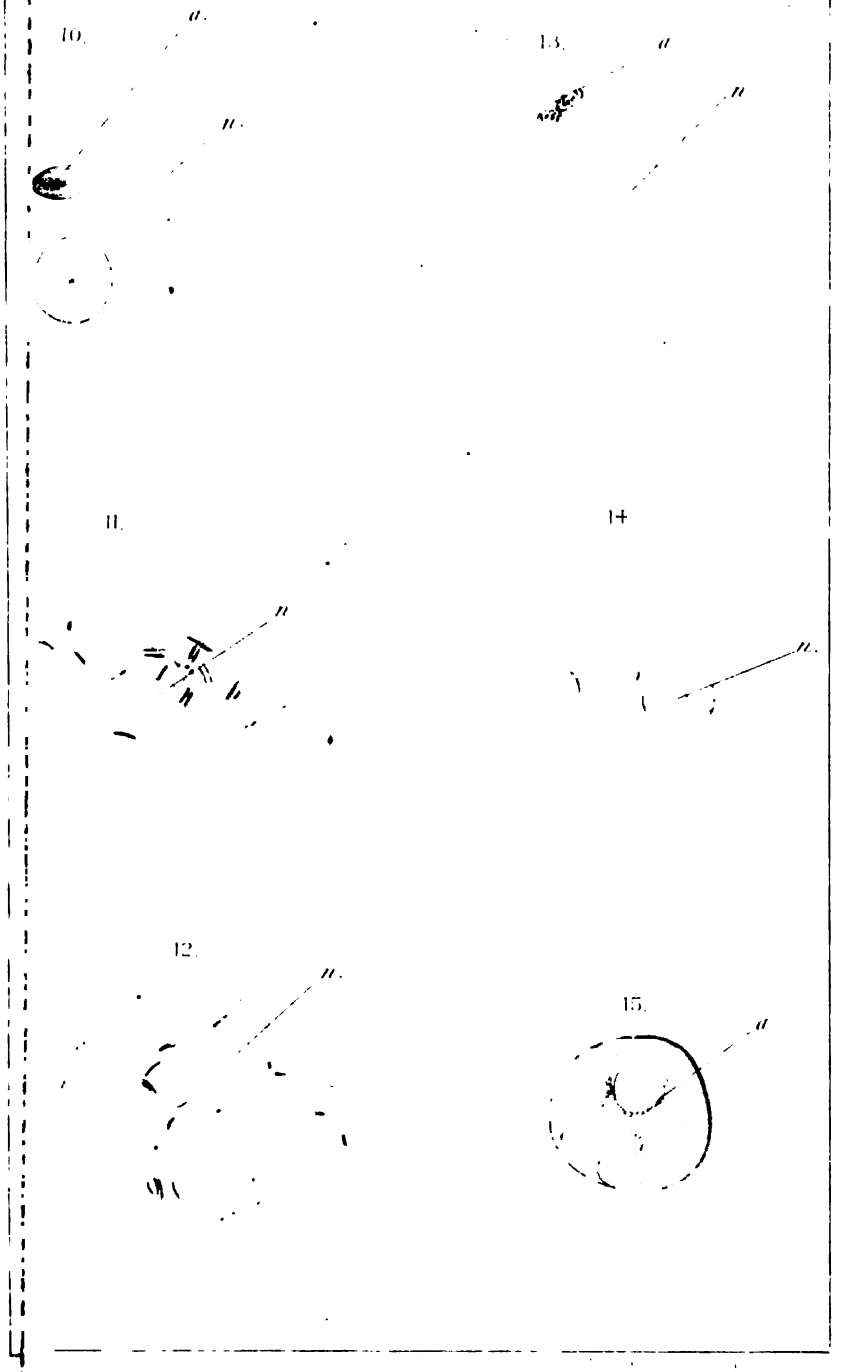

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INDEX TO VOLUME II.

ABDOMINAL neoplasms, effect of laparotomy on, 108;
Abdominal surgery, drainage tube in, 184.
Abscess of lung, 504.
Abscesses and alterations in liver, 490.
Acute angio neurotic œdema, 339; literature of, 349; symptoms of, 339; report of cases of, 342, 344, 346.
Adenitis, gonorrhœal, extirpation of inguinal glands for, 290.
Albers, 536.
Amœba dysenteriæ, description of, 405; movements of, 406; pseudopodia, 406; endosarc, 408.
Amœbæ, in liver abscesses, 502; in liver tissue, 496; methods of studying, 409; multiplication of, in intestine, 489; in dysenteric stools, 459; action of toxic products of, on the liver cells, 510; probable mode of entry into liver, 513; in abscess of lung, 508; in bronchi, 508; probable mode of infection by, 512.
Amœbic dysentery, 395; analysis of symptoms, 455; anæmia, 456; circulation and respiration, 464; classification of cases, 452; clinical cases, 413, 414, 420, 423, 427, 428, 431, 433, 434, 438, 442, 445, 446, 448, 449; complications, 465; diagnosis of, 473; diarrhœa, 456; fever, rigors and sweating, 463; general clinical review, 452; general condition, 455; onset and course of, 454; peritonitis, 473; termination, 455; abdominal symptoms, 462; sensitiveness to pressure, 463; tenesmus, 463; nausea and vomiting, 463; hiccough, 463.
Amputations of the thigh, 284.
Anæmia, acute, pyrodin in, 41.
Ankle joint, excision of, 274.
Annesley, 527, 541, 517, 518, 519.
Anomalies of the uvula, 32.
Antiseptic and aseptic rules in force in the gynecological operating room, 131.
Arthritis, gonorrhœal, incision of joints for, 289.
Arthrodesis, for paralytic flail-joint, 285.
Arthrotomy, for rupture of posterior crucial ligament, 268.
Axillary glands, excision of, 276.

BACTERIA, 509; in liver abscesses, 503.
Baille, 536.
Baley, 519, 529, 542.
Ballingall, 541.
Basch, 526.
Becker, 527.
Bengal dysentery, 519.
Bérenger-Féraud, 522.
Berkley, Dr., case of chorea insaniens, 317.
Blood-clot, value of, in management of dead spaces, 255.
Bone abscesses, necrotomies and operations for, 292.
Bones, operations for syphilis of, 291.
Bougarel, 526, 542.
Bow legs, osteotomy for, 288.
Bronchi, adjoining amœbic abscess, 507.
Bütschli, 412.

CAMBAY, 523.
Cancer of liver, 3.
Carcinoma of breast, operation for, 277.
Carcinoma of the cervix uteri in the negress, 224.

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Cardiac thrombi, case of ball thrombus, 56; case of laminated thrombus, 58; rare forms of, 56.
Carpal bones, operations for dislocation of, 271.
Carswell, 536.
Catarrhal dysentery, stools of, 461.
Catteloup, 541, 526.
Cerebro-spinal syphilis, case of, 369; autopsy of, 372; microscopical examination, 374.
Cervical glands, excision of, 275.
Cheyne, 528, 542.
Chiari, 542.
Chickahominy diarrhœa, 533, 540.
Chisholm, Colin, 517–524.
Chorea insaniens, case of, 319; autopsy, 322; microscopic examination, 324.
Chronic dysentery, stools in, 458.
Chuckerbutty, 541.
Cirrhosis of liver, 4.
Clitoris, elephantiasis of, 227; myxo-sarcoma of, 231.
Composite temperature and pulse charts, in abdominal section cases, 177; in normal cases, 178; in high temperature cases, 180; in pus cases, 182.
Contents of liver abscesses, 491, 492; of lung abscess, 504.
Cornil, 524, 531.
Councilman, Dr., amœbic dysentery, 395; lesions in yellow fever, 499.
Cruveilhier, 536.
Cuneiform bone, operation for subluxation and fracture of, 265.
Cystoma, laparotomy for, 158.
Cystosarcoma of ovary, autopsy, 173.
Cysts, operations for removal of, 296.

Davis, 517.
De Castro, 542.
Dead spaces, 304.
Descriptions of amœbæ, 396; Lambl, 396; Cunningham, 396; Davaine (*cercomonas hominis*), 396; Malmsten (*paramœcium coli*), 396; Cunningham, 396; 399; Lösch, 397; Koch, 399; Kartulis, 400; Grassi, 400; Perroncito, 400; Calandruccio, 400; Blanchard, 400; Leukart, 400; Hlava, 402; Masiutin, 402; Pfeifer, 402; Osler, 402; Lafleur, 403; Simon, 403; Musser, 403; Stengel, 403; Dock, 403; Cahen, 403; Baumgarten, 403; Baelz, 404.
Dickenson, 543.
Diphtheritic dysentery, stools of, 462.
Division of flexor tendons for contracted fingers, 270.
Double uvula, 32; cases of, 33; varieties of, 33.
Drainage tube, in abdominal surgery, 184; in capillary drainage, 185; care of tube, 188; conditions for use of, 184; culture experiments with, 192; description of tube, 185; modes of dressing, 190; position of, 185; removal of, 191.
Dysentery, in Algiers, 521; in America, 536; in Bay View, 538; in Cochin China, 524, 526; in Crimea, 519; in Europe, 526; in Guy's Hospital, 530; in India, 517; in Ireland, 528; in Martinique, 522; in Millbank Prison, 529; in Norway, 522; in Senegal, 522, 523; in U. S. Army, 532.
Dysentery, solitary follicles of intestine in, 520.
Dutroulau, 521.

Elbow, exsection of, 263, 272.
Elephantiasis of clitoris, 227.
Endocarditis in phthisis, 62; post-mortem conditions, 62; tubercle bacillus, 64.
Excision, of ankle-joint, 274; of axillary glands, 276; of cervical glands, 275; of inguinal glands, 276; partial, of knee joint, 273; of supraorbital nerve, 271; of tuberculous lymphomata, 275; of tuberculous ulcer of foot, 276.
Exsection, of elbow, 263; of elbow joint, 272; of head of humerus, 272; of portion of radius and ulna, 268.
Extirpation, of capsule of shoulder-joint, 272; of head of femur, 264; of ingui-

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nal glands for adenitis, 290; of varicose veins, 295.
Eyre, 541.

Fagge, 530.
Fallopian tubes, in tubercular peritonitis, 69; tubercle bacillus in sections of, 201.
Femur, extirpation of head of, 264.
Fever of hepatic origin, etc., 3; cancer in, 3; cirrhosis in, 4; obstruction of duct by gallstones, 4.
Fibroid tumors, laparotomy in, 164.
Finger, 527–542.
Flat foot, Trendelenberg-Hahn operation for, 286.
Flexor tendons, division of, for contracted fingers, 270.
Forms of liver abscess, 492, 501.
Fox, 542.
Fracture of internal cuneiform bone, 265; of neck of femur, 264; of patella, operations for, 287; of radius and ulna, 266.
French, 533.

Gallstones, intermittent fever of, 3; obstruction of duct by, 4.
Gangrenous dysentery, stools in, 457.
Gastric symptoms, in tubercular peritonitis, 73.
Gayme, 542.
Gely, 523, 524.
Ghriskey, Dr., report of urinary examinations in gynecological cases, 216.
Glands, axillary, excision of, 276; cervical, excision of, 275; inguinal, excision of, 276; inguinal, extirpation of, for gonorrhœal adenitis, 290.
Gluge, 536.
Gonococcus, history of case, 196; microscopic examination, 199; in pyosalpinx, 195.
Griesinger, 542.
Gynecological cases, urinary examination of, 216.
Gynecological operations, general, 205; tabulated report of, 208; deaths, 243; methods of perineal operation, 206.
Gynecological operating room, 131; dressings, 139; essentials of, 133; preparation of operator and assistants, 137; of patients, 135; treatment of instruments, 135.

Hæmoglobinuria, in Raynaud's disease, 114.
Hæmatomyelia, 351; differential diagnosis of, 365; cases of, 353, 360.
Halsted, Dr., treatment of wounds by the blood-clot in dead spaces, 255.
Haspel, 522, 523.
Hemorrhage from bowel, 473; from uterus, means of checking, 220.
Hepatic and hepato-pulmonary abscesses, 465; time of development, 466; site, 466; duration, 467; symptoms, 468; fever, 468; rigors and sweating, 468; nausea and vomiting, 469; jaundice, 469; circulation, 469; respiration, 469; cough and expectoration, 469; sputa, 469; microscopical character of, 470; amœbæ, 471; physical signs, 471; auscultation, 472; inspection, 471; palpation, 471; percussion, 472.
Hernia, inguinal operations for radical cure of, 281; umbilical, laparotomy for, 165.
Hewson, 516, 519.
Hip-joint disease, osteotomy for, 267.
Hlava, 542.
Hoch, Dr., hæmatomyelia, 351.
Hope, 536.
Horton, 520.
Humerus, exsection of head of, 272; operation for gumma of, 291; sequestrotomy for osteo-myelitis of, 293.
Hunter, John, 516.
Hysteria, pyrodin in, 43.
Hysterorrhaphy, new method, 163.

Incision and irrigation of joints for gonorrhœal arthritis, 289.
Inflammation of appendages, laparotomy in cases of, 162.

Digitized by Google

Inguinal glands, excision of, 276; hernia, operation for, 281.
Injections of exudate into skin, 302; of staphylococcus into muscles, 301.
Insanity, post-febrile, 46.
Intermittent pyrexia, associated with gall-stones, 3; cases of, 6–20; differential diagnosis, 20; pathology of, 28; prognosis of, 30; treatment, 30.
Intestinal lesions, 477.
Intracapsular fracture of neck of femur, 264.
Irrigation of joints for arthritis, 289.

JULIEN, 526, 542.

KARTULIS, 537.
Kelsch and Kiener, 487, 523, 525, 537.
Kelly, Dr., composite temperature and pulse charts, 177; elephantiasis of the clitoris, 227; general gynecological operations from October 15, 1889, to March 4, 1890, 205; kolpo-ureterotomy, etc., 234; ligature of the trunks of the uterine and ovarian arteries as a means of checking hemorrhage from the uterus, etc., 220; record of deaths following gynecological operations, 243.
Kelly, Dr., the gynecological operating room and the antiseptic and aseptic rules in force, 131; report of the autopsies in two cases dying in the gynecological wards without operation, 167; tuberculosis of the Fallopian tubes and peritoneum, ovarian tumor, 201; the gonococcus in pyosalpinx, 195.
—— and Ghriskey, Dr., report of urinary examination in gynecological cases, 216.
—— and Robb, Dr., laparotomies performed in the gynecological department from October 16, 1889, to March 3, 1890, 141.
Kidneys, changes in amœbic dysentery, 510.
Knee-joint, partial excision of, 273; sarcoma of, 284.
Kolpo-ureterotomy, history of case of, 235; operation for, 240; operation per vaginam, 234.

LAFLEUR, Dr., on pyrodin, 37; amœbic dysentery, 395.
Laparotomies, analysis of cases, 156; for cystomata, 158; effect on abdominal neoplasms, 108; for fibroid tumors, 164; for inflammation of appendages, 162; for ovaritis, 159; for umbilical hernia, 165; tabulated record of cases, 141, 142.
Lesions of solitary follicles in amœbic dysentery, 489.
Lebert, 536.
Leidy, 533.
Leukæmia, pyrodin in, 41.
Ligature of uterine and ovarian arteries, 220.
Lipoma of thorax, removal of, 297.
Liver, abscesses and alterations in, 490; anatomical changes in, 537; contents of, 491, 492; in Egypt, 542, 543; in England, 543; forms of, 492, 501; lesions in, 503; mortality of, 541, 542; walls of, 491, 501.
Liver, cancer of, 3; cirrhosis of, 4.
Liver cells, 493, 494, 500; necrosis of, 496, 498; nuclei of, 497; regeneration of, 499.
Liver tissue, 491, 492, 493.
Lung, abscess of, 504; contents of, 504; adjoining amœbic abscess, 505; changes in alveolar epithelium near amœbic abscess, 511; connective tissue in alveoli, 507.
Lymphomata, tuberculous, excision of, 275.
Lyons, 519, 542.

MACKENZIE, Dr., anomalies of uvula, with special reference to double uvula, 32.
Maclean, 520.
Macpherson, 519, 541.
Malaria, pyrodin in, 38, 39, 40.

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Management of dead spaces in treatment of wounds, 304; disinfection of of hands, 307; dressings, 306; of operation, 308; operating room, 311; preparation of patient, 307; of sutures and ligatures, 305; operating table, 311.
Mesenteric glands, tumors of, in tubercular peritonitis, 88.
Methods of studying amœbæ, 409.
Monroe, 516.
Moracchi, 528.
Moore, 541, 543.
Morehead, 519, 520.
Mouret, 541.
Myoma of uterus, autopsy in case of, 168.
Myxo-sarcoma of the clitoris, 231.

Necrosis of liver cells, 496, 498.
Necrotomies and operations for bone abscesses, 292.
Nephritis, acute, in typhoid, 119; cases illustrating clinical features of, 120, 123; morbid anatomy of, 122, 126.
New growths, operations for removal of, 296.
Nuclei of liver cells, 497.

Obstruction of duct by gallstones, symptoms of, 4; cases of, 6–20; without fever, cases of, 23.
Omental tumors, in tubercular peritonitis, 79.
Operation for carcinoma of breast, 277; for dislocation of carpal bones, 271; for drop wrist, 266; for fractures of patella, 287; for gonorrhœal pyosalpinx, 197; for inguinal hernia, 281; for removal of cysts and new growths, 296; for syphilis of bones, 291; for syphilitic periostitis of rib, 292; for tuberculosis of bones and joints, 272; for ununited fractures, 286.
Orth, 531.
Osborn, 528.
Osler, Dr., acute nephritis in typhoid fever, 119; cases of post-febrile insanity, 46; note on endocarditis in phthisis, 62; on fever of hepatic origin, particularly the intermittent pyrexia associated with gallstones, 3; rare forms of cardiac thrombi, 56; tubercular peritonitis, 67.
Osteoma of scapula, operation for, 296.
Osteotomy, for bow-legs, 288; subtrochanteric for hip-joint disease, 267.
Osteo-myelitis, of humerus, 293; of tibia, sequestrotomy for, 292.
Ovaritis, laparotomy for, 159.

Paralytic flail-joint, arthrodesis for, 285.
Parkes, 519, 520.
Patella, operations for fractures of, 287.
Periostitis, syphilitic, of rib, operation for, 292.
Peritonitis, acute, and tubercular peritonitis, 67, 72; character of, produced by amœbæ, 508; purulent, injection of exudate into skin, 302.
Phthisis, endocarditis in, 62; use of pyrodin in, 37, 38, 41.
Pigmentation, in tubercular peritonitis, 77.
Pleura, involvement of, in tubercular peritonitis, 69.
Pneumonia, and acute tuberculosis, diagnosis, 53; insanity following, 46; pyrodin in, 38.
Post-febrile insanity, 46; in pneumonia, 46; in typhoid, 47, 48, 49.
Pringle, Sir John, 516, 519.
Prognosis of amœbic dysentery, 475.
Pyogenic micro-organisms, exclusion of from wounds, 298.
Pyosalpinx, gonococcus in, 195.
Pyrodin, 37; in acute anæmia and leuksæmia, 41; in hysteria, 43; in malaria, 38, 39, 40; morbid changes in rabbit produced by, 44; in phthisis, 37, 38, 41; in pneumonia, 38; in typhoid fever, 40, 41, 43.

Radical cure of inguinal hernia, operations for, 281.

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Radius, fracture of, 266; exsection of portion of, 268.
Ranking, 541.
Raynaud's disease, 114.
Record of deaths following gynecological operations, 243.
Regeneration of liver cells, 499.
Renal complications in typhoid, 119.
Renal intermittent fever, 29.
Rib, syphilitic periostitis of, operation for, 292.
Robb, Dr., myxo-sarcoma of the clitoris, 231; the management of the drainage tube in abdominal surgery, 184; laparotomies performed in gynecological department, 141.
Rokitansky, 519, 531, 542.

SARCOMA of knee-joint, amputation for, 284.
Scapula, osteoma of, removal, 296.
Sequestrotomy for osteo-myelitis of the humerus, 293; for osteo-myelitis of the tibia, 292.
Shanks, 520.
Shoulder-joint, extirpation of capsule of, 272.
Simon, Dr., acute angio-neurotic œdema, 339.
Solitary follicles, lesions of, 489.
Spinal cord, unusual lesion of, in case of cerebro-spinal syphilis, 369.
Staphylococcus aureus, inoculation of peritoneal cavity with, 301; inoculation of muscles with, 301.
Stools, in gangrenous dysentery, 457; in chronic dysentery, 458; in catarrhal dysentery, 461; microscopic examination of, 458; in diphtheritic dysentery, 462.
Subluxation of internal cuneiform bone, operation for, 265.
Supraorbital nerve, excision of, 271.
Surgical operating room, 311; sutures and ligatures, 305; dressings, 306; disinfection of hands, 307; preparation of patient, 307; operation, 308; operating table, 311.
Syphilis, cerebro-spinal, case of, 369; of bones, operations for, 291.

TENOTOMY for contracted fingers, 270; for hip-joint disease, 267.
Testicle, syphilitic, removal of, 297.
Thigh, amputations of, 284; varicose veins, extirpation of, 295.
Thomas, Dr., 524; case of cerebro-spinal syphilis, with an unusual lesion in the spinal cord, 369; case of Raynaud's disease, associated with convulsions and hæmoglobinuria, 114.
Toulmin, Dr., acute tuberculosis in an infant of four months, 51.
Trendelenberg-Hahn operation for the cure of acquired flat foot, 286.
Tubercle bacillus in sections of Fallopian tube, 201.
Tubercular peritonitis, 67; age in, 69; with gastric symptoms, 73; simulating simple acute peritonitis, 72; classification of, 68; clinical manifestations, 71; cure by operation, 98; curability of, 92; diagnosis of, 90; effect of laparotomy on abdominal neoplasms, 108; general considerations, 67; mesenteric glands, 88; morbid anatomy of, 68; omental tumors, 79; pigmentation, 77; race in, 70; retracted and thickened intestinal coils in, 85; sacculated exudations, 82; sex in, 70; simulating typhoid, 75; spontaneous cure of, 93; subnormal temperature, 77; symptoms of, ascites, 76; tumor formations in, 78; tympanitis, 76.
Tuberculosis, acute, in infant of four months, 51; autopsy, 52; and pneumonia, diagnosis, 53; source of infection, 54.
Tuberculosis, of bones and joints, operations for, 272; pathological report, 204; of the Fallopian tubes and peritoneum, 201; of the Fallopian tubes, removal of tubes and ovaries, 203.
Tuberculous hip-joint disease, operation for, 267.
Tuberculous lymphomata, excision of, 275.

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Tuberculous ulcer of foot, excision of, 276.
Typhoid fever, acute nephritis in, 119; insanity following, 47, 48, 49; pyrodin in, 40, 41, 43; tubercular peritonitis simulating, 75.

ULNA, fracture of, 266; exsection of portion of, 268.
Umbilical hernia, laparotomy for, 165.
U. S. army medical museum, 535.
Urinary examination in gynecological cases, 216.
Urine, in amœbic dysentery, 464.
Ureteral stricture, incision of ureter for, 234.
Uvula, anomalies of, 32.

VARICOSE veins of leg and thigh, extirpation of, 295.
Vogt, 526, 527.

WALCHEREN diseases, 517.
Walls of liver abscess, 491, 501.
Waring, 541.
Williams, Dr., carcinoma of the cervix uteri in the negress, 224.
Wollaston, 516.
Woodward, 532, 534, 535, 517, 542.
Wounds, exclusion of micro-organisms from, 298; experiments with staphyloccus aureus in, 301, 302; summary of "blood-clot" wounds, 262; treatment of, etc., 255.

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